PHASE 2 REPORT- REVIEW COPY
FURTHER SITE CHARACTERIZATION AND ANALYSIS

VOLUME 2F - HUMAN HEALTH RISK ASSESSMENT

HUDSON RIVER PCBs REASSESSMENT RI/FS

AUGUST 1999

For

U.S. Environmental Protection Agency
Region II
and
U.S. Army Corps of Engineers
Kansas City District

Book 1 of 1
Upper Hudson Risk Assessment

TAMS Consultants, Inc.
Gradient Corporation

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

August 4, 1999

To All Interested Parties:

The U.S. Environmental Protection Agency (USEPA) is pleased to release the baseline Human Health Risk Assessment for the Upper Hudson River (HHRA), which is part of Phase 2 of the Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS) for the Hudson River PCBs Superfund site. The HHRA evaluates current and future risk to adults, adolescents, and children posed by PCBs in the Upper Hudson River in the absence of remediation. The HHRA will help establish acceptable exposure levels for use in developing remedial alternatives in the Feasibility Study, which is Phase 3 of the Reassessment RI/FS for the Hudson River PCBs site.

As stated in the April 1999 Responsiveness Summary for Phase 2 - Human Health Risk Assessment Scope of Work, USEPA will complete the Mid-Hudson Human Health Risk Assessment following review of the revised Thomann-Farley model developed for the Hudson River Foundation.

USEPA will accept comments on the HHRA until September 7, 1999. Comments should be marked with the name of the report and should include the report section and page number for each comment. Comments should be sent to:

Alison A. Hess, C.P.G.
USEPA Region 2
290 Broadway - 19th Floor
New York, NY 10007-1866
Attn: Upper Hudson River HHRA Comments

USEPA will hold two Joint Liaison Group meetings to discuss the findings of the HHRA. The first meeting will be on the date of release of the report, August 4, 1999, and will be held at 7:30 p.m. at the Marriott Hotel, 189 Wolf Road, Albany, New York. The second meeting will be on August 5, 1999 at 7:30 p.m. at the Sheraton Hotel, 40 Civic Center Plaza, Poughkeepsie, New York. Both meetings are open to the general public. Notification of these meetings was sent to Liaison Group members, interested parties, and the press several weeks prior to the meetings.

During the public comment period, USEPA will hold availability sessions to answer questions from the public regarding the HHRA. The availability sessions will be held from 2:30 to 4:30 p.m. and from 6:30 to 8:30 p.m. on August 18, 1999 at the Holiday Inn Express, 946 New Loudon Road, Latham, New York.

If you need additional information regarding the HHRA, the availability sessions, or the Reassessment RI/FS in general, please contact Ann Rychlenski, the Community Relations Coordinator for this site, at (212) 637-3672.

Sincerely yours,

William McCabe

Richard L. Caspe, Director
Emergency and Remedial Response Division

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Human Health Risk Assessment: Upper Hudson River
Executive Summary
August 1999

This document presents the baseline Human Health Risk Assessment for the Upper Hudson River (HHRA), which is part of Phase 2 of the Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS) for the Hudson River PCBs site in New York.[1] This HHRA quantitatively evaluates both cancer risks and non-cancer health hazards from exposure to polychlorinated biphenyls (PCBs) in the Upper Hudson River, which extends from Hudson Falls, New York to the Federal Dam at Troy, New York. The HHRA evaluates both current and future risks to children, adolescents, and adults in the absence of any remedial action and institutional controls. The HHRA uses current U.S. Environmental Protection Agency (USEPA) policy and guidance as well as additional site data and analyses to update USEPA's 1991 risk assessment.

USEPA uses risk assessment as a tool to evaluate the likelihood and degree of chemical exposure and the possible adverse health effects associated with such exposure. The basic steps of the Superfund human health risk assessment process are the following: 1) Data Collection and Analysis to determine the nature and extent of chemical contamination in environmental media, such as sediment, water, and fish; 2) Exposure Assessment, which is an identification of possible exposed populations and an estimation of human chemical intake through exposure routes such as ingestion, inhalation, or skin contact; 3) Toxicity Assessment, which is an evaluation of chemical toxicity including cancer and non-cancer health effects from exposure to chemicals; and 4) Risk Characterization, which describes the likelihood and degree of chemical exposure at a site and the possible adverse health effects associated with such exposure.

The HHRA shows that cancer risks and non-cancer health hazards to the reasonably maximally exposed (RME) individual associated with ingestion of PCBs in fish from the Upper Hudson River are above levels of concern. Consistent with USEPA regulations, the risk managers in the Superfund program evaluate the risk and hazards to the RME individual in the decision-making process. The HHRA indicates that fish ingestion represents the primary pathway for PCB exposure and for potential adverse health effects, and that risks from other exposure pathways are generally below levels of concern. The results of the HHRA will help establish acceptable exposure levels for use in developing remedial alternatives for PCB-contaminated sediments in the Upper Hudson River, which is Phase 3 (Feasibility Study) of the Reassessment RI/FS.

Data Collection and Analysis

USEPA previously released reports on the nature and extent of contamination in the Upper Hudson River as part of the Reassessment RI/FS (*e.g.*, February 1997 Data Evaluation and Interpretation Report, July 1998 Low Resolution Sediment Coring Report, August 1998 Database for the Hudson River PCBs Reassessment RI/FS [Release 4.1], and May 1999 Baseline Modeling Report). The Reassessment RI/FS documents provide current and forecasted concentrations of PCBs in fish, sediments, and river water and form the basis of the site data collection and analyses used in conducting the HHRA.

[1] A separate human health risk assessment is being conducted for the Mid-Hudson River (Federal Dam at Troy, New York to Poughkeepsie, New York).

Exposure Assessment

Adults, adolescents, and children were identified as populations possibly exposed to PCBs in the Upper Hudson River due to fishing and recreational activities (swimming, wading), as well as from living adjacent to the Upper Hudson River and inhaling volatilized PCBs in the air. Cancer risks and non-cancer hazards were calculated for each of these populations. To protect human health and provide a full characterization of the PCB risks and hazards, both an average (central tendency) exposure estimate and an RME estimate were calculated. The RME is the maximum exposure that is reasonably expected to occur in the Upper Hudson River under baseline conditions.

The exposure pathways identified in the HHRA are ingestion of fish, incidental ingestion of sediments, dermal contact with sediments and river water, and inhalation of volatilized PCBs in air. For these exposure pathways, central tendency and RME estimates were calculated using point estimate analyses, whereby an individual point estimate was selected for each exposure factor used in the calculations of cancer risks and non-cancer health hazards. Incidental ingestion of river water while swimming was not evaluated because the river water meets drinking water standards for PCBs.

In addition to the point estimate analysis, a Monte Carlo analysis was performed to provide a range of estimates of the cancer risks and non-cancer health hazards associated with the fish ingestion pathway. The Monte Carlo analysis helps evaluate variability in exposure parameters (*e.g.*, differences within a population's fish ingestion rates, number of years an angler is exposed, body weight) and uncertainty (*i.e.*,a lack of complete knowledge about specific variables).

Ingestion of Fish

For fish ingestion, both central tendency and RME estimates were developed for each of the parameters needed to calculate the cancer risks and non-cancer health hazards. Based on the 1991 New York Angler survey of fish consumption by licensed anglers (Connelly *et al.*, 1992), the central tendency fish ingestion rate was determined to be approximately six half-pound meals per year and the RME fish ingestion rate was determined to be 51 half-pound meals per year.

For the point estimate analyses, cancer risks and non-cancer health hazards to an adult angler were calculated. Population mobility data from the U.S. Census Bureau for the five counties surrounding the Upper Hudson River and fishing duration data from the 1991 New York Angler survey were used to determine the length of time an angler fishes in the Upper Hudson River (*i.e.*, exposure duration). The exposure duration for fish ingestion was 12 years for the central tendency exposure estimate and 40 years for cancer (7 years for non-cancer) for the RME estimate. Standard USEPA default factors were used for angler body weight. Future concentrations of PCBs in fish were derived from forecasts presented in the Baseline Modeling Report, which were then grouped by fish species and averaged over species for the entire Upper Hudson River. PCB losses during cooking were assumed to be 20% for the central tendency exposure estimate and 0% (no loss) for the RME estimate, based on studies reported in the scientific literature.

In the Monte Carlo analyses, each exposure parameter (*e.g.*, ingestion rate, exposure duration, body weight) was represented by a range of values, each with an assigned probability, rather than as a single point estimate. Cancer risks and non-cancer hazards were calculated for anglers beginning at age 10. Differences in the length of time an angler fishes the Upper Hudson (exposure duration) were obtained from the 1991 New York Angler survey and the U.S. Census Bureau data. Differences in angler body weight through time were obtained from national health surveys summarized in the scientific literature. Future concentrations of PCBs in fish were

derived from the Baseline Modeling Report. Fish species consumption variability was evaluated based on consumption patterns determined from the 1991 New York Angler survey and within-species PCB concentrations were averaged over location within the Upper Hudson River. The variability in fish ingestion rates was examined by considering surveys of fish ingestion rates in states other than New York. Variability in PCB cooking loss was determined from a review of the scientific literature.

Due to the lack of sufficient information available to define quantitative uncertainty distributions for several important exposure factors, such as exposure duration, an explicit two-dimensional Monte Carlo analysis which examines variability and uncertainty separately could not be performed. Instead, an expanded one-dimensional (1-D) analysis was completed using a sensitivity/uncertainty analysis. Each 1-D Monte Carlo simulation examined variability of PCB intake and was repeated for a range of possible input distributions for important exposure variables. A total of 72 separate combinations of the variable input parameters were examined in the 1-D analysis. Each 1-D simulation consisted of 10,000 simulated anglers, such that the entire 1-D Monte Carlo analysis consisted of 720,000 simulations.

Other Exposure Pathways

For the direct exposure scenarios for river water and sediment, the central tendency exposure estimates for adults and young children (aged 1-6) were assumed to be one day every other week for the 13 weeks of summer (7 days/year) and for the RME were assumed to be one day per week for the 13 weeks of summer (13 days/year). Adolescents (aged 7-18) were assumed to have about three times more frequent exposure, with a central tendency exposure estimate of 20 days/year and an RME estimate of 39 days/year. The risks due to possible inhalation of PCBs in air were evaluated for both recreational users of the river (swimmers and waders) as well as for residents living adjacent to the Upper Hudson River. The concentrations of PCBs in water and sediment were derived from the Baseline Modeling Report. The concentrations of PCBs in air were calculated from a combination of historical monitoring data and modeled emissions from the river using a USEPA-recommended air dispersion model. Standard USEPA default factors were used for certain exposure parameters (*e.g.*, body weight) in the risk calculations for these pathways.

Toxicity Assessment

The toxicity assessment is an evaluation of the chronic (7 years or more) adverse health effects from exposure to PCBs (USEPA, 1989b). In Superfund, two types of adverse health effects are evaluated: 1) the incremental risk of developing cancer due to exposure to chemicals and 2) the hazards associated with non-cancer health effects, such as reproductive impairment, developmental disorders, disruption of specific organ functions, and learning problems. The cancer risk is expressed as a probability and is based on the cancer potency of the chemical, known as a cancer slope factor, or CSF. The non-cancer hazard is expressed as the ratio of the chemical intake (dose) to a Reference Dose, or RfD. The chronic RfD represents an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive populations (*e.g.*, children), that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chemical exposures exceeding the RfD do not predict specific diseases. USEPA's Integrated Risk Information System, known as IRIS, provides the primary database of chemical-specific toxicity information used in Superfund risk assessments. The most current CSFs and RfDs for PCBs were used in calculating cancer risks and non-cancer hazards in the HHRA.

PCBs are a group of synthetic organic chemicals consisting of 209 individual chlorinated biphenyls called congeners. Some PCB congeners are considered to be structurally similar to dioxin and are called dioxin-like PCBs. USEPA has classified PCBs as a probable human carcinogen, based on a number of studies in laboratory

animals showing liver tumors. Human carcinogenicity data for PCB mixtures are limited. USEPA (1996) described three published studies that analyzed deaths from cancer in PCB capacitor manufacturing plants (Bertazzi *et al.*, 1987; Brown, 1987; Sinks *et al.*, 1992). Recently, Kimbrough *et al.* (1999) published the results of an epidemiological study of mortality in workers from two General Electric Company capacitor manufacturing plants in New York State. Due to the limitations of the Kimbrough *et al.* (1999) study identified by USEPA in its review (*e.g.*, more than 75% of the workers never worked with PCBs, the median exposure for those who worked with PCBs was only a few years, and the level of PCB exposure could not be confirmed), USEPA expects that the study will not lead to any change in its CSFs for PCBs, which were last reassessed in 1996.

Risk Characterization

Point Estimate Calculations

Ingestion of fish contaminated with PCBs resulted in the highest lifetime cancer risks. The RME estimate of the increased risk of an individual developing cancer averaged over a lifetime based on the exposure assumptions is 1×10^{-3}, or one additional case of cancer in 1,000 exposed people. The RME risks associated with the dioxin-like PCBs are comparable. The central tendency (average) estimate of risk is 3×10^{-5}, or 3 additional cases of cancer in 100,000 exposed people. For known or suspected carcinogens, acceptable exposure levels for Superfund are generally concentration levels that represent an incremental upper bound lifetime cancer risk to an RME individual of between 10^{-4} and 10^{-6}. The central tendency cancer risks and non-cancer hazards are provided to more fully describe the health effects associated with average exposure. Estimated cancer risks relating to PCB exposure in sediment and water while swimming or wading, or from inhalation of volatilized PCBs in air by residents living near the river, are much lower than those for fish ingestion, falling generally at the low end, or below, the range of 10^{-4} to 10^{-6}. A summary of the point estimate cancer risk calculations is presented below.

Point Estimate Cancer Risk Summary		
Pathway	**Central Tendency Risk**	**RME Risk**
Ingestion of Fish	3×10^{-5} (3 in 100,000)	1×10^{-3} (1 in 1,000)
Exposure to Sediment*	4×10^{-7} (4 in 10,000,000)	1×10^{-5} (1 in 100,000)
Exposure to Water*	1×10^{-8} (1 in 100,000,000)	2×10^{-7} (2 in 10,000,000)
Inhalation of Air*	2×10^{-8} (2 in 100,000,000)	1×10^{-6} (1 in 1,000,000)

**Total risk for child (aged 1-6), adolescent (aged 7-18), and adult (over 18).*

The evaluation of non-cancer health effects involved comparing the average daily exposure levels (dose) to determine whether the estimated exposures exceed the Reference Dose (RfD). The ratio of the site-specific calculated dose to the RfD for each exposure pathway is summed to calculate the Hazard Index (HI) for the exposed individual. An HI of one (1) is the reference level established by USEPA above which concerns about non-cancer health effects must be evaluated.

Ingestion of fish resulted in the highest Hazard Indices, with an HI of 10 for the central tendency point

estimate and an HI of 116 for the RME point estimate. The total HIs for exposure to sediment, water, and air are all below one. Non-cancer hazards due to inhalation of PCBs were not calculated because IRIS does not contain a toxicity value for inhalation of PCBs. A summary of the point estimate non-cancer hazards is presented below.

Point Estimate Non-Cancer Hazard Summary		
Pathway	**Central Tendency Non-Cancer Hazard Index**	**RME Non-Cancer Hazard Index**
Ingestion of Fish	10	116
Exposure to Sediment*	0.05	0.2
Exposure to Water*	0.007	0.02
Inhalation of Air	Not Calculated	Not Calculated

**Values for child and adolescent, which are higher than adult for these pathways.*

Monte Carlo Estimate

In the Monte Carlo analysis, a distribution of cancer risks and non-cancer health hazards was calculated for the fish ingestion pathway. The tables below summarize the low-end (5th percentile), midpoint (50th percentile), and high-end ($\geq$ 90th percentile) cancer risks and non-cancer hazards. At a given percentile, the risks or hazards are higher than that presented in the table for 100 minus the given percentile. For example, as shown for the base case in the table below, the calculated incremental cancer risk at the 95th percentile is 9 x 10^{-4}, which means that the cancer risks for only the top 5th percentile are greater than that value.

Monte Carlo Cancer Risk Summary - Fish Ingestion			
Risk Percentile	**Low Estimate**	**Base Case**	**High Estimate**
5th Percentile	7×10^{-7}	5×10^{-6}	5×10^{-5}
50th Percentile	1×10^{-5}	6×10^{-5}	4×10^{-4}
90th Percentile	7×10^{-5}	5×10^{-4}	2×10^{-3}
95th Percentile	1×10^{-4}	9×10^{-4}	3×10^{-3}
99th Percentile	3×10^{-4}	4×10^{-3}	1×10^{-2}

Monte Carlo Non-Cancer Hazard Summary - Fish Ingestion			
Risk Percentile	**Low Estimate**	**Base Case**	**High Estimate**
5th Percentile	0.1	1	7
50th Percentile	2	11	51
90th Percentile	5	31	117
95th Percentile	11	82	233
99th Percentile	19	136	366

Comparison of Point Estimate and Monte Carlo Analyses

The Monte Carlo base case scenario is the one from which point estimate exposure factors for fish ingestion were drawn, thus the point estimate RMEs and the Monte Carlo base case estimates are comparable. Similarly, the point estimate central tendency (average) and the Monte Carlo base case midpoint (50th percentile) are comparable. For cancer risk, the point estimate RME for fish ingestion (1×10^{-3}) falls approximately at the 95th percentile from the Monte Carlo base case analysis. The point estimate central tendency value (3×10^{-5}) and the Monte Carlo base case 50th percentile value (6×10^{-5}) are similar. For non-cancer hazards, the point estimate RME for fish ingestion (116) falls between the 95th and 99th percentiles of the Monte Carlo base case. The point estimate central tendency HI (10) is approximately equal to the 50th percentile of the Monte Carlo base case HI of 11.

Major Findings of the HHRA

The HHRA evaluated both cancer risks and non-cancer health hazards to children, adolescents and adults posed by PCBs in the Upper Hudson River. USEPA has classified PCBs as probable human carcinogens and known animal carcinogens. Other long-term adverse health effects of PCBs observed in laboratory animals include a reduced ability to fight infections, low birth weights, and learning problems. The major findings of the report are:

- Eating fish is the primary pathway for humans to be exposed to PCBs from the Hudson.
- Under the RME scenario for eating fish, the calculated risk is one additional case of cancer for every 1,000 people exposed. This excess cancer risk is 1,000 times higher than USEPA's goal of protection and ten times higher than the highest risk level allowed under Superfund law.
- For non-cancer health effects, the RME scenario for eating fish from the Upper Hudson results in a level of exposure to PCBs that is more than 100 times higher than USEPA's reference level (Hazard Index) of one.
- Under the baseline conditions, the point estimate RME cancer risks and non-cancer hazards would be above USEPA's generally acceptable levels for a 40-year exposure period beginning in 1999.
- Risks from being exposed to PCBs in the river through skin contact with contaminated sediments and river water, incidental ingestion of sediments, and inhalation of PCBs in air are generally within or below USEPA's levels of concern.

1 Overview of Upper Hudson River Risk Assessment

1.1 Introduction

This document presents the baseline Human Health Risk Assessment (HHRA) for the Upper Hudson River as required under the National Oil and Hazardous Substances Pollution Contingency Plan (USEPA, 1990). This assessment quantifies both carcinogenic and non-carcinogenic health effects from exposure to polychlorinated biphenyls (PCBs) in the Upper Hudson River, following USEPA risk assessment policies and guidance. This assessment evaluates both current and future risks to children, adolescents and adults based on the assumption of no remediation or institutional controls (USEPA, 1990).

The risk assessment considers site data collected during the late 1970s and early 1980s, and data collected during the Reassessment Remedial Investigation and Feasibility Study (RI/FS) which started in 1990. This assessment relies primarily on data from the Phase 2 Investigation contained in the database for the Hudson River PCBs Reassessment RI/FS,[1] as summarized in the following documents: the Database Report (USEPA, 1995a); the Preliminary Model Calibration Report (USEPA, 1996a); the Data Evaluation and Interpretation Report (USEPA, 1997d); and the Baseline Modeling Report (USEPA, 1999d).

1.2 Site Background

The Hudson River PCBs Superfund Site extends from Hudson Falls, NY to the Battery (at the southern tip of Manhattan) in New York City. The site covers approximately 200 river miles. Specifically, as stated in the USEPA's April 1984 Feasibility Study:

> The environment affected by the Hudson River PCB problem includes all waters, lands, ecosystems, communities and facilities located in or immediately adjacent to the 200-mile stretch of river from Fort Edward to the Battery. This project focuses on, *but is not limited to*, the most heavily contaminated reach between Albany and Fort Edward (Upper Hudson River) (emphasis added). (1984 Feasibility Study at ES-4).

Similarly, in the USEPA's September 25, 1984 Record of Decision (ROD), the site is defined by reference to three figures which, together, depict the Site as the entire 200-mile stretch of the River from Hudson Falls to the Battery in New York City, plus the remnant deposits. This HHRA addresses the Upper Hudson River, which is the area between Hudson Falls, NY and the Federal Dam in Troy, NY, a length of approximately 40 river miles (Plate 1).[2]

From 1957 through 1975, between 209,000 and 1,300,000 pounds of PCBs were discharged to the Upper Hudson River from two General Electric facilities: one located in Fort Edward, NY and the other

[1] Database for the Hudson River PCBs Reassessment RI/FS, Release 4.1b, August 1998.

[2] A separate risk assessment is being conducted using similar methodologies for the Mid-Hudson River (the area between Federal Dam in Troy, NY and Poughkeepsie, NY), a length of approximately 83 river miles. The Mid-Hudson analysis will be presented upon the completion of USEPA's review of the appropriateness of the PCB bioaccumulation modeling for the Lower Hudson River that is being conducted under a grant from the Hudson River Foundation to Drs. Thomann and Farley.

in Hudson Falls, NY (USEPA, 1991a). In 1977, the manufacture processing and distribution commerce of PCBs within the U.S. was restricted under provisions of the Toxic Substances and Control Act (USEPA, 1978).

In 1973, the Fort Edward Dam was removed, which facilitated the downstream movement of PCB-contaminated sediments (USEPA, 1991a). Because of potential human health risks due to consumption of PCB-contaminated fish, the New York State Department of Environmental Conservation (NYSDEC) and the New York State Department of Health (NYSDOH) banned fishing in the Upper Hudson River and limited the recommended number of fish meals consumed for specific species in the Lower Hudson River (NYSDOH, 1995). In 1976, the commercial striped bass fishery in the Hudson River was closed based on elevated PCB levels in striped bass. The ban on fishing in the Upper Hudson River was subsequently changed to a "catch and release" program in August 1996, however advisories against consumption of any fish from the Upper Hudson River remain in effect (NYSDOH, 1999).

In 1984, USEPA issued a ROD for the site. The ROD required: 1) an interim No Action decision concerning river sediments; 2) in-place capping, containment and monitoring of remnant deposit sediments; and 3) a treatability study to evaluate the effectiveness of removing PCBs from the Hudson River water (USEPA, 1984).

1.3 General Risk Assessment Process

The goal of the Superfund human health evaluation process is to provide a framework for developing the risk information necessary to assist in the determination of possible remedial actions at a site. USEPA uses risk assessment as a tool to characterize the contaminants, evaluate the toxicity of the chemicals, assess the potential ways in which an individual may be exposed to the contaminants, and characterize the cancer risks and non-cancer hazards (USEPA, 1989b). In accordance with USEPA guidance, actions at Superfund sites are based on an estimate of the reasonable maximum exposure (RME) expected to occur under both current and future conditions at the site. The RME is defined as the highest exposure that is reasonably expected to occur at a site. USEPA guidance also recommends the Agency estimate risks based on central tendency, or average, exposures at a site (USEPA, 1995b). The RME and central tendency exposures are used to estimate cancer risks and non-cancer health hazards.

A systematic framework for human health assessment was first outlined in 1983 by the National Academy of Sciences (NRC, 1983). Building upon that foundation, the risk assessment process described in USEPA's "Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A)" (USEPA, 1989b) and subsequent Agency guidance consists of the following components:

- *Data Collection and Analysis* - involves gathering data, including the use of models as necessary, to define the nature and extent of contamination.

- *Exposure Assessment* - entails an estimate of the magnitude of actual and/or potential human exposures, the frequency and duration of these exposures, and the pathways (*i.e.*, inhalation, ingestion, and dermal contact) by which people are potentially exposed.

- *Toxicity Assessment* - examines the type of adverse health effects associated with chemical exposure, and the relationship of the magnitude of exposure and the health response.

- *Risk Characterization* - summarizes the results from the first three steps of the assessment (both quantitative and qualitative) and a discussion of the uncertainties in the analysis.

The data collection and analysis step in the risk assessment process has been documented at length in other Phase 1 and Phase 2 Reassessment RI/FS reports. The HHRA draws upon those data and analyses, and provides the reader with references to relevant reports where a description of the information used in this HHRA can be found in greater detail.

1.4 Discussion of 1991 Phase 1 Risk Assessment

In 1991, USEPA issued the Phase 1 Report - Interim Characterization and Evaluation for the Hudson River PCB Reassessment Remedial Investigation/Feasibility Study, including a quantitative risk assessment for the Upper Hudson River and a qualitative risk assessment for the Lower Hudson River (USEPA, 1991a). The Phase 1 Risk Assessment identified potential cancer risks and non-cancer hazards associated with regular consumption of fish from the Upper Hudson River exceeding guidelines established in the NCP for acceptable risk.

The Phase 1 Upper Hudson River human health risk assessment evaluated current and potential future risks from ingestion of fish, ingestion of drinking water, dermal contact with sediments, dermal contact with river water, and incidental ingestion of sediments. A map of the Upper Hudson River study area is shown in Plate 1.

The cancer risks from ingestion of fish were 2×10^{-2} (*i.e.*, an excess cancer risk of 2 in a population of 100) using the 1986-1988 95% Upper Confidence Limit on the Mean (95% UCLM) PCB concentration in fish (12.0 mg/kg), and 2×10^{-3} using the 30-year projected mean PCB concentration in fish (1.5 mg/kg) (USEPA, 1991a). The non-cancer Hazard Index for ingestion of fish was 51 using the 1986-1988 95% UCLM PCB concentration, and 6 using the 30-year projected mean PCB concentration in fish.

As described in the NCP (USEPA, 1990), "For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} to 10^{-6} using information on the relationship between dose and response." The cancer risks calculated in Phase 1 exceeded the range defined in the NCP; the non-cancer Hazard Index exceeded one (1), indicating an exceedance of the Reference Dose, or the level at which no adverse chronic health effects are expected to occur.

The cancer risk from drinking water was 6×10^{-6}, within the acceptable risk range defined in the NCP, and the non-cancer Hazard Index was less than one (USEPA, 1991a). Cancer risks from dermal exposure to river sediment, incidental ingestion of river sediment, and dermal contact with river water totaled 8.8×10^{-6}, also within the acceptable risk range, and the non-cancer Hazard Index was also less

than one (USEPA, 1991a). Risks from other pathways including ingestion of vegetables and meat, and inhalation exposures were evaluated qualitatively in the Phase 1 risk assessment.

The Phase 1 Lower Hudson River human health risk assessment qualitatively evaluated current and potential risks from ingestion of fish, based on the findings in the Upper Hudson River. The assessment concluded that the risks from ingestion of fish would be similar to those found in the Upper Hudson River. A human health risk assessment for the Mid-Hudson River will be presented upon the review and determination of the appropriateness of the Farley-Thomann model of PCB bioaccumulation in fish species of the Mid- and Lower Hudson.

1.5 Objectives of Phase 2 Risk Assessment

In December 1990, USEPA Region 2 began a reassessment of the No-Action decision for the Hudson River sediments based on, among other things, a request by NYSDEC and requirements of the Superfund Amendments and Reauthorization Act of 1986 to conduct reviews every five years of remedial decisions for sites where contamination remains on site. The reassessment consists of three phases: interim characterization and evaluation; further site characterization and analysis; and a Feasibility Study. As part of the Phase 2 Reassessment, this report presents the Human Health Risk Assessment for the Upper Hudson River. An ecological risk assessment for the Hudson River is also being completed.

Since the Phase 1 Risk Assessment, there have been additional data and information compiled that are incorporated into this Phase 2 assessment:

- An extensive amount of additional PCB data have been collected in water, sediment, fish and other biota.
- PCB concentration trends in environmental media have been forecast using extensive modeling efforts.
- An extensive review of fish ingestion surveys was conducted to determine the most appropriate fish ingestion rate for the HHRA.
- The cancer toxicity of PCBs has undergone an extensive review by USEPA and the scientific community resulting in updated toxicity factors for PCBs, and the revised toxicity values for PCBs are lower than those in effect when the Phase 1 assessment was completed based on new animal studies and revisions in USEPA's cancer guidelines. A reassessment of PCB non-cancer toxicity is underway.

The objectives of the Phase 2 risk assessment are to update the findings from Phase 1 (that risks from fish ingestion outweigh other pathways of exposure), taking into consideration the additional information highlighted above, and to provide estimates of risks both to the RME, or high-end risk estimates (>90^{th} to 99^{th} percentiles), as well as estimates of risks to the Average Exposed Individual, or central tendency risk estimates (50^{th} percentile). This HHRA is limited to evaluating potential health risks associated with PCBs, because the HHRA is being conducted as part of USEPA's Reassessment of its 1984 No-Action decision for the PCB-contaminated sediments in the Upper Hudson River.

2 Exposure Assessment

The objective of the exposure assessment is to estimate the magnitude of human exposure to PCBs in the study area. USEPA guidance (USEPA, 1989a,b; 1991b; 1992a,b,c; 1995b; 1996b; 1997a,e,f) provides the framework adopted to conduct the exposure assessment for this risk assessment.

The population of concern in this HHRA consists of the inhabitants of the towns, cities, and rural areas surrounding the Upper Hudson River who may fish or engage in activities that will bring them into contact with the river. In the discussion that follows, certain terms used by risk assessors are introduced to define specific subgroups of this population. For example, members of the population who fish are described as the "angler" population. In addition, specific types of activities (*e.g.*, recreation) give rise to the use of the term "recreator" to describe another possible segment of the exposed population. The term "receptor" or "receptor population" is used to describe these subgroups of the exposed population. This definition of several receptor population groups does not suggest that these represent distinct individuals or even separate populations. Thus, individuals in the population of concern may fall within each of the "angler," "recreator," and "resident" receptor groups described below and throughout this HHRA. Distinguishing separate receptor groups does not imply these populations are mutually exclusive, but rather the receptor groups are defined for convenience of distinguishing different PCB exposure possibilities.

Human exposures to PCBs in the environment are quantified by determining the concentration of PCBs in environmental media (air, water, sediment, fish) which humans may then ingest or otherwise contact resulting in PCB uptake into the body. The exposure assessment process involves determining the concentration of PCBs in the environmental media of concern, and combining this information with estimates of human exposure to the environmental media. The variability of environmental concentrations, the likelihood of exposure occurring *via* particular pathways, and the frequency and duration of human exposure are all components of the analysis.

USEPA guidance and policy call for an evaluation of a central estimate of risk, and an estimate of risk for a reasonable maximum exposure, or RME, individual. An estimate of the RME can be obtained by determining estimates of likely "high-end" exposure factors and then combining these high-end factors with average factors to come up with a point estimate, or single value, for the reasonable maximum exposure. Alternatively, the RME can be estimated using probabilistic methods, often involving a technique termed Monte Carlo analysis (USEPA, 1997a). Such a Monte Carlo analysis does not estimate the RME based on single point estimates for each exposure factor, but rather draws repeated plausible exposure factor values from a probability distribution characterizing each factor, and combines these repeated samples to develop a distribution of exposure estimates. This distribution of PCB exposure contains an explicit estimate of the probability associated with any particular PCB exposure (intake) estimate, such that the RME can be determined based on estimates from the high-end of the Monte Carlo exposure distributions.

In this HHRA, point estimates of exposure (and cancer risk and non-cancer hazard) are developed for both central tendency and RME exposures for all exposure pathways that are considered to be complete (see next section). This point estimate method is the same as the approach adopted in the Phase 1 risk assessment, taking into consideration the important new information outlined in Section 1.5, and is described in the Risk Assessment Guidance for Superfund - Part A (USEPA, 1989b). In addition, a Monte Carlo exposure analysis is conducted for the fish ingestion pathway, the pathway shown in the

Phase 1 risk assessment to yield the highest exposure to PCBs. For clarity, the point estimate exposure analysis is presented in this chapter (Chapter 2) of the report. The Monte Carlo exposure analysis for the fish ingestion pathway is presented in Chapter 3. Because some of the point estimate exposure factors (*e.g.*, fish ingestion rate, exposure duration, *etc*.) are based upon the sources of information and probability distributions for these factors derived in Chapter 3, the reader is referred to the Monte Carlo analysis for further details on these exposure factors where they are discussed more fully.

Section 2.1 summarizes the environmental media, potential receptors, and exposure pathways of PCB intake for the HHRA. The framework for calculating human intake resulting from PCB exposures is presented in Section 2.2. The PCB exposure point concentrations used to estimate PCB intake are summarized in Section 2.3. Finally, the exposure factors and algorithms used to calculate PCB intake, and estimates of PCB intake for each complete exposure pathway, are summarized in Section 2.4. In this report, exposure assessment information is tabulated in USEPA's *Risk Assessment Guidance for Superfund* (RAGS), Part D format (USEPA, 1997e) in order to promote consistency of presenting risk assessment information to the public.

2.1 Exposure Pathways

For exposure and potential risks to occur, a complete exposure pathway must exist. A complete pathway requires the following elements (USEPA, 1989a):

- A source and mechanism for release of constituents,
- A transport or retention medium,
- A point of potential human contact (exposure point) with the affected medium, and
- An exposure route (*e.g.*, ingestion, dermal contact, inhalation) at the exposure point.

If any one of these elements is missing, the pathway is not considered complete. For example, if human activity patterns and/or the location of potentially exposed individuals relative to the location of affected media prevents human contact, then that exposure pathway is not complete and there is no health risk in such instances. Considering the sources of PCBs, potential release mechanisms, likely exposure media, potential receptors, and possible intake mechanisms, the complete exposure pathways at the site were identified. The exposure scenarios examined in this HHRA assume no remediation and no institutional controls that would limit environmental exposures.

The Upper Hudson River study area for this HHRA includes urban, suburban, and rural areas along the river. During boating, fishing, and other recreational activities members of the Upper Hudson River study area population may become exposed to PCBs if they consume fish caught from the river, or as they come into contact with river water and river sediments; they could also inhale PCBs that may be released from the water into the air. Potential exposure pathways considered in this HHRA are summarized in Table 2-1, identifying those which are "complete" and warranted exposure and risk calculations in this study. The following sections describe site-specific elements that make up the complete exposure pathways that are evaluated in this HHRA.

2.1.1 Potential Exposure Media

Humans may be exposed to PCBs from the site either through direct ingestion or contact with media containing PCBs. In addition, PCB exposure can result from the transfer of PCBs from one medium (water) to another (air). PCBs have been detected, monitored and modeled extensively at the site. The exposure media that are considered the most potentially significant source of PCB exposure at the site include the following:

Fish. Fish bioaccumulate PCBs, and as the results of the Phase 1 risk assessment indicate, ingestion of fish is likely to be the predominant pathway for human exposure to PCBs in the Upper Hudson River.

Sediment. Swimming, wading, and boating along the Hudson are recreational activities that would likely give rise to contact with sediment. Therefore, sediment is a potential exposure medium at the site.

River Water. Similar to river sediment, exposure to surface water from the Upper Hudson River is likely to occur during recreational activities and river water is thus considered a potential exposure medium.

Air. PCBs that volatilize from the river water may be inhaled by both recreators and residents living near the river. This medium is being considered in this assessment in order to update information presented in the Phase 1 risk assessment and address concerns raised by the public regarding potential inhalation of PCBs.

The actual determination of the relative importance of each of these potential exposure media, and those which may or may not pose a significant health risk, is determined based on the results of the quantitative exposure and risk analysis.

2.1.2 Potential Receptors

As described in the opening of this section, the population of concern in the evaluation of the Upper Hudson River consists of the inhabitants of the towns, cities, and rural areas surrounding the river. From this population, the following "receptor" groups have been defined for the purpose of quantifying the potential PCB exposures within the population as a whole. As indicated at the outset of this chapter, these receptor groups should not be interpreted as though they represent distinct population subgroups, rather they are defined for convenience of presenting the exposure and risk analysis.

Anglers. The analysis from the Phase 1 Report (USEPA, 1991a) revealed that estimated PCB intake through consumption of fish from the Hudson River is the most significant pathway of human exposures to PCBs at the site; therefore, much of the effort for the HHRA is focused on refining the estimates of PCB exposure to anglers. The angler population is defined as those individuals who consume self-caught fish from the Hudson, in the absence of a fishing ban or Hudson-specific health advisories. The assessment of fish consumption by the angler population includes childhood through adulthood.

Fishing is an increasingly popular recreational activity. In 1988, an estimated 26,870 anglers fished on the Hudson River; of those, an estimated 10,310 fished specifically on the Upper Hudson River (Connelly *et al.*, 1990). Based on the estimated number of angler days over time, angling effort in the state of New York appears to be increasing over time (Jackson, 1990).

Recreators. Recreators along the Upper Hudson River are another potential receptor population group defined in this HHRA This receptor population includes individuals participating in recreational activities along the river such as swimming, wading, boating, picnicking, *etc*. Because recreational activity patterns change with the age of the population, exposure by young children (aged 1-6), older children and teenagers (aged 7-18), and adults (aged 18 and above) are considered separately.

Residents. Although both of the above receptor groups include residents of the Upper Hudson River study area, a third receptor group, termed "residents," has been assigned for the purpose of assessing long-term exposure to PCB-contaminated air for that portion of the population living in close proximity to the river.

2.1.3 Potential Exposure Routes

An exposure route is the means, or mechanism, of contact with an exposure medium. Typical routes of exposure include dietary intake, inadvertent or incidental ingestion or intake of environmental media, air inhalation, *etc*. For anglers in the Upper Hudson River area, fish ingestion (*e.g.*, dietary intake) is the potential exposure route evaluated in this risk assessment. Routes of exposure under a recreational use scenario include absorption of PCBs *via* dermal contact with sediments, incidental ingestion of PCBs contained in sediments during subsequent hand to mouth contact, dermal contact with river water, and inhalation of air. Ingestion of river water was not quantitatively evaluated in this risk assessment because this exposure route was found to have *de minimis* risk, using reasonable maximum assumptions, in the Phase 1 assessment (USEPA, 1991a). Furthermore, the current, and projected future, PCB concentrations in the Upper Hudson River are below the drinking water maximum contaminant level (MCL). Inhalation of air is also a potential exposure route for residents who live in close proximity to the Upper Hudson River. Each of these exposure routes is summarized in Table 2-1.

In addition to the above-mentioned routes of exposure, other potential pathways exist by which individuals may be exposed to PCBs originating from the Upper Hudson River. Such pathways include dietary intake of home-grown crops, and consumption of local beef or dairy products. Although insufficient data exist to provide a detailed quantitative analysis of these exposure pathways, the discussion below indicates they are unlikely to be a significant pathway for PCB intake.

For the last 25 years, the New York State Department of Agriculture and Markets has analyzed more than 18,200 samples of cow's milk within the state and has not found any detection of PCBs above the detection limit of 0.6 ppm (lipid normalized).[3] Moreover, in the 1980s, Dr. Buckley from the Boyce Thompson Institute at Cornell University collected data on PCBs in forage crops (corn and hay) grown in an area with PCB-contaminated soil. He found that levels of PCBs on these crops (sources of animal

[3] This detection limit is significantly less than the FDA limit of 1.5 ppm (lipid normalized) (FDA, 1996).

food) were below the U.S. Department of Agriculture regulatory level of 0.2 mg/kg for forage crops. Based on this information, the risk *via* ingestion from foods other than Hudson River fish is likely to be minimal, and collection of additional PCB data from vegetables, meat, eggs and milk is not warranted.

In addition, a few snapping turtles in the Upper Hudson River have been found to contain PCBs (Stone *et al.*, 1980; Olafsson *et al.*, 1983). Because of the small number of turtles that have been analyzed, the data may not be representative. Furthermore, it is also unknown whether turtles are caught and consumed by local residents. Consumption of fish is considered to be a more likely important dietary pathway for PCB intake from the Upper Hudson River. Thus, the overall intake from possible occasional consumption of other non-fish biota (such as turtles) would likely result in lower PCB intake estimates than those quantitatively evaluated here for fish based on the frequency and duration of exposure.

2.2 Quantification of Exposure

In this section of the risk assessment, the basic approach for calculating human intake levels resulting from exposures to PCBs is presented. Exposure estimates represent the daily dose of a chemical taken into the body, averaged over the appropriate exposure period. Chemical intake is expressed in terms of a dose, having units of milligram chemical per kilogram body weight per day (mg/kg-day). In general, quantitative exposure estimates involve the following:

- determination of exposure point concentrations (the concentration of PCBs in environmental media at the point of human exposure);
- identification of applicable human exposure models and input parameters (exposure frequency, duration, *etc*.); and
- estimation of human intakes using exposure algorithms.

The primary source for the exposure algorithms used in the risk assessment is USEPA's Risk Assessment Guidance for Superfund, Part A (RAGS) (USEPA, 1989b). The generalized equation for calculating chemical intakes is:

$$I = \frac{C \times CR \times EF \times ED \times CF}{BW \times AT}$$

where:

I	=	*Intake* - the amount of chemical at the exchange boundary (mg/kg body weight/day)
C	=	*Exposure Point Concentration* - the chemical concentration contacted over the exposure period at the exposure point (*e.g.*, mg/kg-fish)
CR	=	*Contact Rate* - the amount of affected medium contacted per unit time or event (*e.g.*, fish ingestion rate in g/day)
EF	=	*Exposure frequency* - describes how often exposure occurs (days/year)
ED	=	*Exposure duration* - describes how long exposure occurs (yr)

CF = *Conversion factor* - (kg/g)

BW = *Body weight* - the average body weight over the exposure period (kg)

AT = *Averaging time* - period over which exposure is averaged (days)

Exposure parameters (*e.g.*, contact rate, exposure frequency, exposure duration, body weight) describe the exposure of a receptor for a given exposure scenario. These values are the input parameters for the exposure algorithms used to estimate chemical intake (USEPA, 1989b; USEPA, 1991b; USEPA, 1997f). The general equation above is slightly modified for each pathway, and the specific exposure parameters for each pathway are summarized and discussed in detail in Section 2.4.

For each of the potentially complete exposure pathways identified in Table 2-1, both central and RME exposure estimates are calculated in this HHRA. The RME is the maximum exposure that is reasonably expected to occur at the site (USEPA, 1989b). A combination of Agency-recommended values and site-specific values were used for each of the input parameters. According to USEPA guidance (1995b), central tendency estimates are intended to reflect central estimates of exposure or dose, while RME estimates are intended to reflect persons at the upper end ("above about the 90^{th} percentile") of the distribution. RME, or high-end, exposure estimates should be within the range of possible exposures, and not beyond.

High-end risk descriptors, according to USEPA (1995b), are defined as "plausible estimates of the individual risk for those persons at the upper end of the risk distribution." When a sufficient database is available, USEPA (1995b) recommends reporting exposures "at a set of selected percentiles of the distributions, such as 90^{th}, 95^{th}, and 98^{th} percentile." The use of the 90^{th} to 95^{th} percentile estimates of exposure parameters for the high end exposure assessment for the Upper Hudson River study area is consistent with this guidance, and reflects the upper range of exposures, but not necessarily the maximum possible exposure.

2.3 Exposure Point Concentrations

A typical baseline Superfund risk assessment includes an evaluation of those chemicals at a contaminated site that pose a potential health concern, or chemicals of potential concern (COPCs). In this HHRA PCBs are identified as the COPCs, because this HHRA is being conducted as part of USEPA's Reassessment of its 1984 No-Action decision for the PCB-contaminated sediments in the Upper Hudson River. Consequently, no screening of COPCs was performed for this assessment. Thus, the USEPA RAGS Part D format tables (Tables 2-2 through 2-5) which for a typical risk assessment would include information necessary to determine COPCs, are not needed and are included in this HHRA only for consistency.

Another consideration which shapes the determination of the exposure point concentrations (EPC) in this HHRA is the time- and space-dependency of the PCB concentrations in fish, sediment, and water. Moreover, the EPC for PCBs in each of these media is based upon modeled projections of future concentrations in each medium (although the models are based upon a large monitoring record). As a result, the typical approach adopted in Superfund risk assessments of calculating an upper confidence limit on a mean concentration (*i.e.*, 95% UCLM), in some instances no longer strictly applies. One reason for its inapplicability is that the 95% UCLM calculation is based upon the notion that the estimate of the mean

exposure point concentration from a finite sample set is uncertain and is a function of the number of samples available to estimate the true mean. However, when a model is used to predict the EPC there is no corollary to sample size; with a model an almost unlimited number of model-predicted values can be calculated. As the number of model-projected concentration estimates increases (in time or space), the model mean and model 95% UCLM converge to the same value. Only if model inputs are varied to reflect environmental variability of the model input parameters, and repeated model estimates of the mean are obtained over the range of parameters, can an average and 95% upper confidence limit on the modeled means be calculated.

2.3.1 PCB Concentration in Fish

Because the HHRA examines current and future health risks, and because the concentration of PCBs in fish changes over time and location, the EPC for PCBs in fish necessarily relies upon model predictions. Three factors have an influence on the exposure point concentration in fish:

1. The concentration of PCBs for any particular species varies for a particular year, but overall it declines over time.

2. The concentration of PCBs within the same fish species varies with location in the Upper Hudson River, with higher concentrations upstream (Thompson Island Pool) compared to downstream.

3. The concentration of PCBs varies among different fish species.

Thus, even though fish are considered a single exposure medium for the HHRA, each of the above factors will influence the calculation of a single exposure point concentration.

Summary of Modeled PCB Concentration Results

The 1999 report, "Further Site Characterization and Analysis Volume 2D - Baseline Modeling Report" presents a detailed discussion of the PCB bioaccumulation and transport and fate models that have been used by USEPA to predict future trends in PCB concentration in fish (USEPA, 1999d). Several bioaccumulation models were used, one of which adopted an empirical prediction of bioaccumulation based on a bi-variate correlation analysis of PCB concentrations in sediment and the water column with those measured in fish. Another analysis involved a mechanistic food web model, a modification of the Gobas model described as FISHRAND in the Baseline Modeling Report, that used the historical measurements of PCBs in fish, water, and sediment in order to calibrate the model to fish species in the Upper Hudson River. In both cases, the bioaccumulation models rely upon predictions of future PCB concentrations in the water column and sediments (from the HUDTOX model) to predict future trends of PCB concentration in fish. The bioaccumulation models in the Baseline Modeling Report will be externally peer-reviewed along with the entire Baseline Modeling Report. In this HHRA, the FISHRAND model predictions were used to estimate EPCs for fish (USEPA, 1999d).

As described in the Baseline Modeling Report, the fish bioaccumulation models used the extensive database that was created to support the Hudson River PCBs Reassessment RI/FS to calibrate the

models (USEPA, 1995a). The database contains measurements for sediments, fish and aquatic biota, surface water flow and surface water quality from the USEPA, the NYSDEC and General Electric Company. The database includes a total of approximately 750,000 records. Almost 350,000 of these records contain data acquired as part of the USEPA's Phase 2 sampling effort. The remaining records contain data from a large number of historical and ongoing monitoring efforts in the Hudson River. The reader is referred to the Baseline Modeling Report (USEPA, 1999d) for further information on the bioaccumulation and transport and fate models.

Model predictions were provided for six fish species: brown bullhead, largemouth bass, white perch, yellow perch, pumpkinseed, and spottail shiner. These species were selected in the Baseline Modeling Report to get a representative distribution of bottom feeders, species at the top of the food chain, and semi-piscivorous species (USEPA, 1999d). Model estimates of Total PCB concentration in each species were based all PCB congeners with three or more chlorine molecules, *i.e.*, Tri+ PCB concentrations (USEPA, 1999d). For the larger fish species modeled (*i.e.*, brown bullhead, largemouth bass, white perch, and yellow perch), the model provides estimates of PCB concentration in fish fillets, otherwise the model results are for whole fish for the smaller species. The fillet represents the portion of the fish most commonly consumed.

Modeled predictions of future PCB concentrations in fish are presented in the Baseline Modeling Report at four locations in the Upper Hudson River: Thompson Island Pool (approximately River Mile 189); Stillwater Dam (approximately River Mile 168); Waterford (approximately River Mile 157); and near the Federal Dam (approximately River Mile 154). These four locations correspond to locations where an extensive number of fish have been monitored by the NYSDEC. Because of their close proximity, the model predictions at the Waterford and Federal Dam locations were combined to result in approximately equal weighting of the concentration results within the Upper Hudson River.[4] Overall, the concentrations for all fish species decrease with river mile, with concentrations around the Thompson Island Pool being the highest.

The Baseline Modeling Report model yielded estimates of the 50^{th} percentile (median) and 95^{th} upper percentile predictions of annualized PCB concentration in fish at each location. Because environmental concentration data are by definition positive and typically exhibit a positive skew toward larger values, a lognormal distribution often is used to describe such data (USEPA, 1992c). Under the assumption of lognormality, the two modeled percentiles are sufficient to calculate the mean annualized PCB concentration in each species at each location.

In the Baseline Modeling Report (USEPA, 1999d), PCB concentration in fish were modeled from 1984 to 2018. The model forecast (1998 - 2018) period of 20 years was selected in the Baseline Modeling Report because it yielded a forecast time-frame comparable to the approximately 20-year historical monitoring record for the Upper Hudson River. In the HHRA, the assessment period covers present (1999) and future exposure to PCBs that are consumed in fish. Furthermore, the exposure duration for the HHRA extends beyond the 20-year forecast period, up to 40 years for the RME duration, and 70 years for the Monte Carlo analysis (see later sections). In order to extend the 20-year modeled PCB concentration trends to the longer time-frame required for the HHRA, the mean concentration data were plotted over time for each location (Thompson Island Pool, Stillwater, and the average of

[4] If the Waterford and Federal Dam results were treated independently, this would result in increased weighting of the results for the lower stretch of the river compared to the upper stretch of the river.

Waterford/Federal Dam) and each species. An exponential trend/regression line was fit to the historical and modeled annual PCB concentration means to extrapolate the concentration data to the year 2069 (for a potential 70-year exposure duration) for each of the species and locations. While this extrapolation introduces some uncertainty in the estimation of the long-term trend in fish concentration, the correlation coefficients for all cases were 0.95, or larger, indicating a good fit to the data.

Figures 2-1 through 2-9 display the concentration trend over time and location for each of the 3 modeled species used in the HHRA. Note that several modeled species (spottail shiner, pumpkinseed, and white perch) were not included in the HHRA. In the case of the shiner and pumpkinseed, they are small fish and not typically consumed by humans and were modeled in the Baseline Modeling Report as one component of the fish food web that contributes to PCB accumulation higher up in the food chain. White perch are not commonly found in the Upper Hudson River, so they are not included in the HHRA (Trina vonStackelberg, 1999 personal communication), although white perch will be included in the Mid-Hudson risk assessment.

As noted above, the model predictions include the 50^{th} and 95^{th} percentile annualized concentration. These percentiles represent percentiles of the entire distribution of PCB concentration ranges within species, and not the range or uncertainty of the mean concentration in fish. Although a mean concentration can be computed from the two percentiles provided in the Baseline Modeling Report, it is insufficient to provide an estimate of the upper confidence limit on the mean, or 95% UCLM, PCB concentration. As the summary below illustrates, the average ratio of the model predicted 95^{th} percentile is a factor of 2- to 3-fold greater than the 50^{th} percentile concentration (the maximum ratios for each species are nearly identical to their average ratios). Given this modest spread of concentration from the 50^{th} to 95^{th} percentile of the entire distribution, the 95% UCLM concentration would not be expected to be significantly greater than the mean concentration. In this HHRA, the modeled mean concentration of PCBs was used for the EPC in fish.

Average Ratio of 95^{th} Percentile and 50^{th} Percentile Modeled PCB Concentration in Fish

Modeled Fish Species	***Thompson Island Pool***	***Stillwater***	***Waterford/ Federal Dam***
Bullhead	3.4	2.4	2.2
Largemouth Bass	3.4	1.7	1.8
Yellow Perch	3.4	2.1	2.4

Source: Based on model predictions from Baseline Model Report (USEPA, 1999d).

Concentration Averaged Over Locations

With the exception of some limited information in the NYSDOH 1996 study of Hudson River anglers (NYSDOH, 1999), there is insufficient information to quantify fishing preference or frequency at specific locations within the Upper Hudson River. Consequently, projected PCB concentrations in fish were averaged over the three locations that were modeled (the Waterford/Troy Dam locations were pre-averaged and treated as a single location). This averaging essentially presumes a uniform likelihood of

fishing at any location within the Upper Hudson River study area. A sensitivity analysis is included in the HHRA to examine how the exposure and risk estimates vary with fishing location. The sensitivity analysis is presented in Chapter 5.

The PCB concentrations, averaged over location, for each of the modeled species are summarized in Figure 2-10. Modeled PCB concentrations for brown bullhead are the highest; the modeled PCB concentration in largemouth bass and yellow perch are comparable to one another. As documented in the Baseline Modeling Report, the PCB concentration in the spottail shiner and pumpkinseed species had the lowest predicted PCB concentrations of all modeled species; modeled PCB uptake in white perch was comparable to the PCB uptake in brown bullhead (USEPA, 1999d).

PCB Concentration Weighted by Species-Consumption Fractions

In order to take into account the species individuals actually eat from the Upper Hudson River, species-specific intake patterns, derived from the 1991 New York Angler survey (Connelly *et al.*, 1992), were used to calculate the concentration of PCBs ingested in fish. That is, each species of fish has a characteristic PCB concentration, and the effective concentration an angler consumes will be based on the relative percent of different fish species consumed.

A complete discussion of the 1991 New York Angler survey is found in Chapter 3. A summary of the Connelly *et al*. (1992) survey results is provided in Table 3-3, and is described briefly here. A total of 9 specific species, plus a tenth category denoted "other," were included in the Connelly *et al*. (1992) survey. Of the 9 species in the survey, salmon and trout are not commonly found in the Upper Hudson River study area. In addition, very few catfish (there is a separate category for bullhead) were caught in the 1991/2 and 1996 creel surveys of Hudson River anglers (NYSDOH, 1999). Therefore, salmon, trout and catfish, along with the unidentified "other" category, were excluded when determining species ingestion weights. The six species from the Connelly *et al*. (1992) survey that are potentially caught and eaten in the Upper Hudson River, were grouped such that species for which predicted PCB concentrations are unavailable were assigned the PCB concentration of a modeled species that fell within the same group.

Table 3-4 summarizes species-group intake percentages by summing the frequency percentage of the individual species in each group. Fish listed in Group 1, such as the brown bullhead, tend to remain at the bottom of lakes, rivers, and streams for a large portion of their life cycle. In Group 2, bass[5] and walleye are predatory fish, preying on other fish, and can be very large, reaching several feet in length. Perch is the only fish species in Group 3. Using this grouping of fish, the modeled concentrations for the brown bullhead serve as surrogate for the PCB concentration for all Group 1 species; the largemouth bass for all Group 2 species, the yellow perch for Group 3.

The point estimate PCB concentrations were derived using the species ingestion fractions shown in Table 3-4 multiplied by the PCB concentrations in each of the three modeled fish species. Thus, the point estimate of the weighted EPC is:

$$EPC = EPC_{Group1} \times 0.44 + EPC_{Group2} \times 0.47 + EPC_{Group3} \times 0.09$$

[5] The Connelly *et al.* (1992) survey did not specify what specific species were included in "bass." Presumably, this category includes both largemouth and smallmouth bass. The category may include striped bass, and other bass species as well.

The EPC values for fish are summarized in Tables 2-6 through 2-8 for each of the three modeled locations. An overall EPC for the entire Upper Hudson River was calculated by averaging over the three locations. As summarized in Table 2-12, the central tendency EPC of 4.4 mg/kg PCBs was calculated by averaging the species-weighted concentration distribution over the 50^{th} percentile exposure duration estimate (*i.e.*, 12 years). The high-end exposure EPC of 2.2 mg/kg PCBs was calculated by averaging the species-weighted concentration distribution over the 95^{th} percentile exposure duration estimate (*i.e.*, 40 years). The determination of these particular exposure durations is described in Section 2.4.1. and Section 3.2.4.

It may be counter-intuitive that the high-end EPC is lower than the central tendency EPC. This fact is a direct result of the declining PCB concentration in fish. Due to this decline over time, the average concentration over the 40-year exposure duration is less than the average concentration over the 12-year period. However, the total lifetime PCB dose, which combines concentration, exposure duration, and other intake factors, is greater for the high-end (RME) point estimate.

2.3.2 PCB Concentration in Sediment

Just as is the case for fish, PCB concentrations in sediment in the Upper Hudson River change as a function of location and time. In the Baseline Modeling Report (USEPA, 1999d), PCB concentrations in surficial (0 - 4 cm) sediment were modeled over time and distance under two boundary condition scenarios: 1) assuming a zero-upstream source of PCBs, and 2) assuming a constant-upstream source of PCBs. For each scenario the model predictions included Total PCBs and Tri+ PCBs (USEPA, 1999d). The predicted Total PCB concentrations assuming a constant-upstream boundary condition (*i.e.*, assuming a constant source of PCBs to the river sediments) were used to calculate exposure point concentrations.

The model predictions were presented for 10 different river mile segments from Fort Edward (River Mile 195) to the Federal Dam (River Mile 154). Model predictions from the Baseline Modeling Report were differentiated into cohesive and non-cohesive sediment classes for each river segment. The area of cohesive sediment zones is 2.4×10^6 m^2, and the area of non-cohesive sediment is 12.7×10^6 m^2. A plot of the 20-year modeled Total PCB concentrations in sediment is shown in Figure 2-11a. This figure plots the model predictions weighted by the percent of cohesive and non-cohesive sediment in each of the 10 model segments. Because the model segments for the sediment modeling were not uniform, the modeled concentrations were also examined on an area-weighted basis, shown by the lower curve on Figure 2-11a.

In Figure 2-11b, the modeled results for cohesive and non-cohesive sediments are plotted by the River Mile segments. Each point on this plot essentially represents an area averaged concentration (*e.g.*, the model segments yield concentration results that apply over the entire segment modeled). As this figure shows, there is little difference in the modeled PCB concentration for cohesive and non-cohesive sediments. The 20-year average over all plotted River Miles for cohesive sediment is 13.5 mg/kg PCBs, whereas for non-cohesive sediments the average is 15.6 mg/kg PCBs. Given the fact that the two modeled sediment classes do not differ substantially in their PCB concentration, there is no reason to choose sediments from one class or another as the representative sediment class that humans may be exposed to. Thus, the cohesive and non-cohesive classes, weighted by their respective percentages in each model segment, were combined for this HHRA.

It is instructive also to examine Figures 5-4(A-D) of the Baseline Modeling Report (USEPA, 1999d). These figures indicate that the cohesive sediment classes tend to occur in areas along the margins of the river channel, or in areas that may approximate near-shore areas where human contact might be most frequent. However, as just discussed, the PCB concentration in the cohesive sediment class is in fact not appreciably different than the PCB concentration in non-cohesive sediments, and in fact is somewhat lower than the average in non-cohesive sediments. Furthermore, the non-cohesive sediments predominate on a total area basis, even in near-shore areas of the river.

The exposure point concentrations in sediment were calculated from the cohesive/non-cohesive model results by averaging the 20-year results for each of the 10 model segments. Again, given the relatively large scale of the model segments (on the order of one mile to several miles), these 10 segment values represent average concentrations over the entire segment. The mean of these segment averages (14.9 mg/kg PCBs) was used as the central tendency point estimate EPC; the 95th percentile of the 10 segment averages (28.7 mg/kg PCBs) was used as the RME point estimate (Table 2-9). Given the fact that the predictions by segment themselves represent an average over the segment, the 95th upper percentile of these segment predictions can be interpreted as an approximate upper confidence limit on the mean concentration in sediment within the Upper Hudson River exposure unit.

Note that the PCB concentration in sediment was not extrapolated beyond the 20-year model period (as was done for fish). Had the concentrations been extrapolated, the EPCs would decrease, although the decrease would be modest as shown by the relatively flat decline in area-weighted concentration trend shown in Figure 2-11a.

2.3.3 PCB Concentration in River Water

Similar to the sediment results, the Baseline Modeling Report provides model estimated PCB concentrations in the water column over time and distance under two boundary condition scenarios: 1) assuming a zero-upstream source of PCBs, and 2) assuming a constant-upstream source of PCBs. For each scenario the model predictions included Total PCBs and Tri+ PCBs (USEPA, 1999d). The predicted Total PCB concentrations assuming a constant-upstream boundary condition (*i.e.*, assuming a constant source of PCBs to the river sediments) were used to calculate exposure point concentrations.

The water column model predictions from the Baseline Modeling Report segmented the Upper Hudson River into 47 river segments from Fort Edward (River Mile 195) to the Federal Dam (River Mile 154). In some instances (*e.g.*, around islands), the model domain was split into multiple segments that correspond to the same River Mile. In these instances, the PCB concentrations were averaged over the model segments to yield a single concentration value corresponding to the associated River Mile. Of the 47 total model segments, 29 distinct River Miles are represented in the model domain. The 20-year average PCB concentration for each of these 29 River Mile segments is plotted in Figure 2-12a. An indication of the time trend of the model predictions is shown in Figures 2-12b and 2-12c. These figures plot the modeled PCB concentrations over time (model output is on a daily basis) at two particular locations, one at the Thompson Island Dam, and another at Stillwater Dam (note the PCB concentration axis is plotted on a logarithmic scale). As these figures illustrate, there is an overall decline in the predictions over the 20 year period, however the decline is modest. No extrapolation of the water column results beyond the 20-year model period was performed.

As was discussed above for the sediment model results, the water column results represent concentrations over a model segment, or in other words each prediction is an average for the entire model segment. The model segments range from approximately 1/3 mile in length up to approximately 4 miles in length. The 20-year average of the 29 individual River Mile predictions, 2.4×10^{-5} mg/L PCBs (24 ng/L), was used as the central tendency point estimate EPC; the 95th percentile of these 29 predictions, 3.1×10^{-5} mg/L PCBs (31 ng/L), was used in the RME point estimate (Table 2-10). Because the 95th percentile is an upper-bound of a concentration that represents an average over the various model segments, it can be interpreted as an approximate upper confidence limit on the mean concentration in river water within the Upper Hudson River exposure unit.

2.3.4 PCB Concentration in Air

The Phase 1 Report (USEPA, 1991a) provides a discussion of a number of studies that have documented PCB measurements in air in the Upper Hudson River study area, and elsewhere in the State of New York. A wide range of PCB concentrations in air are reported for the general study area, with values measured in the early to late 1980s generally exhibiting concentrations in air on the order of 0.1 $\mu g/m^3$, or less (c.f., Table B.3-21 of Phase 1 Report).

In order to evaluate potential PCB exposure *via* inhalation, the source of the PCBs in air must be linked to the site (*i.e.*, the Upper Hudson River for this HHRA). Although the available air studies indicate PCBs do exist in the atmosphere in the study area, the studies do not necessarily identify the contribution of PCBs in the air that is derived from PCB-contaminated river water.

In order to evaluate the potential quantitative PCB exposure *via* inhalation that is associated with potential releases from the Hudson, three avenues of inquiry were pursued:

1. Historical measurements in 1980-81 of PCBs released to the air from the Hudson near Lock 6 were examined (Buckley and Tofflemire, 1983).

2. The results of the 1991 air monitoring study conducted during remediation of the PCBs in the Remnant Deposit sediments near Fort Edward (released subsequent to the Phase 1 Report) were evaluated.

3. PCB releases from the water column were estimated using diffusion and volatilization equations.

Buckley and Tofflemire 1980-81 Study

Airborne PCB concentrations were monitored at two locations above the Lock 6 dam during the period of 1980-81 (Buckley and Tofflemire, 1983). The location of these monitoring sites was chosen by the authors to represent areas anticipated to have elevated airborne PCB concentrations, owing to the turbulence of the water in the dam spillway which promotes air exchange and increased volatilization potential.

A total of seven samples were taken at a height of 1 meter, and two samples were taken at a height of 4.5 meters. Table A-1 (Appendix A) summarizes the PCB concentrations measured at two locations (A and B) above the Lock 6 dam. Results of Aroclor-specific concentrations for each sample time were summed to get a Total PCB value, assigning one-half the detection limit to non-detected values. Summing all Aroclors to estimate Total PCBs likely overstates the Total PCB concentration. Given the small sample size and historical nature of the results, no adjustment was attempted that would correct for possible overestimating the Total PCB concentration.

Aroclor 1242 was detected in all samples. The Total PCB concentration ranged from 0.033 $\mu g/m^3$ to 0.530 $\mu g/m^3$. The highest detected value may be an outlier result, and was described by the authors as "atypical." The mean of the nine samples is 0.11 $\mu g/m^3$.

Although this study provides evidence suggesting PCBs in air could be attributed to releases from the water column, the study results cannot be used directly to assess current and future potential exposure to PCBs in this HHRA. The results cannot be used because the PCB concentration in the water column (*i.e.*, the source term for the releases from water) was much greater in 1980-81 than current, and projected future, concentrations.

Remnant Deposit Remediation Air Monitoring 1991

As part of the Remnant Deposit Remediation monitoring, Harza Engineering performed air monitoring studies from January through November 1991 (Harza, 1992). The first five months of the monitoring program focused on two miles of the Hudson River in the Fort Edward area and monitored PCB concentrations in air during construction containment activities. After containment was achieved, the remaining monitoring program (June through November 1991) shifted to the Remnant Sites for the first six weeks and then to residential areas for the remainder of the program. Between June and mid-July, one sampler operated on, or adjacent to, each Remnant Site; from mid-July to the end of November, three fixed-location stations (A2, A3, and A4) operated in residential areas (Harza, 1992). Concurrent with the air monitoring, PCBs were monitored in the Hudson River water column.

Overall, 985 airborne PCB samples were collected during the 1991 construction monitoring period. Of these samples, only 13 samples, or 1.3%, had PCB concentrations above the limit of quantification. PCB concentrations (only Aroclor 1242 was detected in 1991) ranging from 0.03 to 0.13 $\mu g/m^3$ were detected during this monitoring program. Table A-2 (Appendix A) presents all detected air sampling results and corresponding river water samples collected in the same vicinity and approximately the same time as the detected air sample results.

A number of factors suggest the PCBs detected in air were emanating largely from the Hudson River, and less likely from the four Remnant Sites or other sources. First, all PCB levels were below the detection limit throughout the first four months of 1991 when the construction containment activities were occurring, and such activities would tend to promote airborne releases of PCBs. Second, the surfaces of the Remnant Sites were covered when these detections occurred (Harza, 1992). Third, PCBs were detected in air only when high PCB concentrations were detected in the water column samples.

These data can be used to estimate an empirical water to air transfer coefficient, representing the ratio of the PCB concentration in air divided by the PCB concentration in water. Using the detected PCB

concentrations in air and water summarized in Table A-2, empirical air-water transfer coefficients range from 0.02 to 0.4 ($\mu g/m^3$ per $\mu g/L$), with a median value of 0.09, and an average value of 0.15 ($\mu g/m^3$ per $\mu g/L$).

According to widely used transport equations used to estimate volatile release of chemicals to air (see discussion of modeling below), at equilibrium, the chemical release to the air is linearly proportional to the chemical concentration in water. Using this principle, the empirical transfer coefficients provide one means of estimating the PCB concentration in air that corresponds to the predictions of future PCB concentrations in the water column. As discussed earlier, the mean predicted PCB concentration in the water column is 24 ng/L (0.024 $\mu g/L$). Applying the median empirical transfer coefficient (0.09), an empirical estimate of the PCB concentration in air associated with an average 0.024 $\mu g/L$ in the water column is 0.002 $\mu g/m^3$. A high-end estimate of the PCB concentration in air, based on the 95th percentile estimate of the water column PCB concentration of 0.042 $\mu g/L$ and the highest empirical transfer coefficient of 0.4, is 0.017 $\mu g/m^3$.

Modeled PCB Concentrations in Air

Another assessment of PCB releases from the Upper Hudson River involved using published modeling approaches, summarized more fully in Appendix A. As described in the Appendix, two approaches were used to estimate the PCB flux from the river. One approach is based on the commonly used two-layer film resistance model as described in Achman *et al.* (1993) and Bopp (1983), and other standard texts. This model describes the volatilization of chemicals as a process of chemical diffusion through a water boundary layer on the water-side of the air-water interface, volatilization at the interface, then diffusion through the air boundary layer on the air-side of the interface. As described in Appendix A, the PCB flux using this model is linearly proportional to the PCB concentration in water, yielding a "normalized" flux rate (mass of chemical per unit concentration in water). Using physical-chemical parameters determined by Bopp (1983) for tri- and tetrachlorobiphenyls, the normalized PCB flux rate is estimated to be:

Normalized PCB Flux (two-film model): 2.7×10^{-3} (ng/m^2-sec per ng/L)

A number of field studies have been conducted examining the flux of PCBs from water bodies to the atmosphere (Nelson *et al.*, 1998; Hornbuckle *et al.*, 1994, Achman *et al.*, 1993; Hornbuckle *et al.*, 1993). Given the complexity of the physical processes controlling the volatilization flux, the estimates using the two-film resistance model were compared with field measurements conducted by Achman *et al.* (1993) in Lake Michigan. Based upon field measurements from June through October, 1989, Achman *et al.* measured the flux of PCBs on 14 separate days, under a range of field conditions (temperature, wind speed, *etc.*). The Total PCB concentration in water measured during the study period ranged from 0.35 ng/L to 7.8 ng/L; measured PCB flux rates ranged from 13 to 1,300 ng/m^2-day (1.5×10^{-4} to 1.5×10^{-2} ng/m^2-sec). The average normalized PCB flux rate (based on the 14 measurements) was:

Normalized PCB Flux (empirical): 1.2×10^{-3} (ng/m^2-sec per ng/L)

The modeled flux rate using the physical-chemical parameters from Bopp (1983) and the empirical PCB flux rate estimates compare favorably. The two-film model estimate is used in the following discussion to estimate the PCB concentration in air in the immediate vicinity of the Upper Hudson River.

The PCB emission estimates provided the PCB source term for the Industrial Source Complex (ISC) air dispersion model (USEPA, 1995c) that was used to estimate PCB concentrations in air in the vicinity of the Upper Hudson River. The ISC model is recommended as a preferred model by the USEPA for use in regulatory and permitting applications. The ISC model was developed by USEPA for determining atmospheric pollutant concentrations associated with point, line, area and volume sources of emission.

Two separate versions of the ISC model are available to allow analysis of both long-term and short-term air quality impacts. The primary difference between the two models is the type of weather data needed as input. The short-term version, ISCST, was designed to calculate contaminant concentrations over time periods as short as one hour. The ISCST model can be used to calculate ambient concentrations over longer time periods (for example one year), simply by averaging the hourly predictions over the appropriate averaging period. Because the ISCST predictions are based upon more detailed meteorologic inputs, the predictions from the ISCST model are considered more accurate than those estimated using the ISCLT (long-term) model. For the HHRA, the current ISC Short Term model, ISCST3 Version 97363 (USEPA, 1995c as updated), was used to estimate the concentration of PCBs in the vicinity of the Upper Hudson River.

As described in Appendix A, a one kilometer (1,000 meter) stretch of river, with an approximate width of 200 meters (a typical width in the Thompson Island Pool area), was modeled.[6] Using the projected average PCB concentration in the Upper Hudson River of 24 ng/L (described earlier) and the normalized flux of 2.7×10^{-3} ng/m^2 per ng/L, the PCB flux estimate for the modeled source area (1000 m $\times$ 200 m) is 13 μg/sec.

The exposure point concentration estimate for PCBs in air depends greatly on the distance from the river. The normalized average downwind PCB concentration modeled using ISCST is estimated to be approximately 70 pg/m^3 per μg/sec at the immediate river edge (downwind), and drop by 10-fold within 200 meters downwind. The average concentration within 50 to 200 meters of the river shoreline is 9 pg/m^3 per μg/sec (Appendix A).

Using the PCB flux just described (13 μg/sec), and the normalized average concentration within 200 meters of shore (9 pg/m^3 per μg/sec), gives a PCB concentration in air of 117 pg/m^3, or 0.00012 μg/m^3. For comparison, if the empirical estimate of PCB flux from the Lake Michigan study (Achman *et al.*, 1993) were used (1.2×10^{-3} ng/m^2-sec per ng/L), the predicted PCB concentration in air within the region 50 to 200 meters from the river shoreline would be 0.00005 μg/m^3.

Estimated Exposure Point Concentration in Air

In summary, there are limited data available that provide site-specific information necessary to estimate future PCB concentrations in air that are attributable to PCB releases from the river. Based on

[6] It should be noted that it is not necessary to model the entire Upper Hudson River. Given the general north-south orientation of the River, the model results are very stable in the east-west direction. Had a longer stretch of river been modeled, the PCB emission rate would have been scaled to the appropriate increase in surface area. The PCB flux per unit area (which is the term that drives the dispersion model), remains constant.

the foregoing discussion, the following range of PCB concentrations in the air for locations near the river that can be reasonably linked to releases from the water column:

Measurements (1980-81):	0.11 $\mu g/m^3$ (mean) 0.53 $\mu g/m^3$ (maximum)
Measurements (1991):	0.03 $\mu g/m^3$ (minimum detected) 0.13 $\mu g/m^3$ (maximum detected)
Empirical Estimate: (1991 Remnant Monitoring)	0.002 $\mu g/m^3$ (central est.) 0.017 $\mu g/m^3$ (high-end est.)
Modeled Estimates:	0.00012 $\mu g/m^3$ (mean water column source) 0.00021 $\mu g/m^3$ (high-end water column source)

The 1980-81 air measurements cannot be used to assess potential current and future PCB exposures because PCB concentrations in the water column were much greater in 1980-81 than current and projected future concentrations. Similarly, to the extent the detected concentration range of PCBs in air measured in 1991 are associated with releases from the water column, the water column PCB concentrations were between one and two orders of magnitude higher in 1991 than they are predicted to be for 1999 - 2020. Thus, using the 1991 measurements directly would likely significantly overstate the airborne PCB concentrations.

Overall, the modeled estimate of PCB concentration in air yield the lowest estimated airborne PCB concentrations. Of the two steps in the air model (first determining the flux rate of PCBs from the water column then using this flux in the ISCST model), modeling the flux rate is the most uncertain. The diffusion coefficients in the flux model are highly dependent on the degree of turbulence in the water column, especially at the air-water interface. The measured flux rates from the Lake Michigan study could be expected to underpredict flux from the Hudson River, which is a flowing, more turbulent, water body. Yet, even if the Lake Michigan flux rates were increased by as much as an order of magnitude, the predicted PCB concentration in air would be 0.0005 $\mu g/m^3$.

Notwithstanding the large range of airborne concentration estimates, a central estimate EPC of 0.001 $\mu g/m^3$ was estimated as the midpoint between the modeled concentration (0.00012 $\mu g/m^3$) and the empirical transfer coefficient estimate (0.002 $\mu g/m^3$). For the RME value, the high-end empirical transfer coefficient estimate of 0.017 $\mu g/m^3$ was chosen as the EPC. These values are summarized in Table 2-11.

2.4 Chemical Intake Algorithms

The following sections describe the calculation of PCB intake for each complete exposure pathway for the HHRA, including the algorithms and exposure parameters. Complete tabulations of the exposure factors for each exposure pathway and receptor scenario are found in Tables 2-12 through 2-24.

2.4.1 Ingestion of Fish

As has been noted earlier, both point estimate and Monte Carlo exposure estimates of PCB exposure *via* fish ingestion are contained in this HHRA. For the point estimate calculations, the intake and risks are calculated for an adult angler, who is likely to ingest the greatest amount a fish over an extended period of time. In the Monte Carlo assessment, the angler population includes fish consumption from childhood through adulthood (Chapter 3). This section summarizes the exposure calculations and factors for the point estimate analysis. Because many of the point estimate factors are based upon the analysis and derivation of their respective probability distributions, which are derived in Chapter 3, the reader is referred to the more complete discussion contained there.

The fish ingestion point estimate intake is calculated as:

$$\text{Intake}_{\text{fish}}\,(\text{mg}/\text{kg}\text{-d}) = \frac{C_{\text{fish}} \times \text{IR} \times (1-\text{LOSS}) \times \text{FS} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

where:

C_{fish}	=	Concentration of PCBs in fish (mg/kg)
IR	=	Annualized fish ingestion rate (g/day)
LOSS	=	Cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion Factor (10^{-3} kg/g)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Table 2-12. Site-specific considerations in selecting these factors are discussed below.

Fraction from Source (FS). This HHRA examines possible exposure for the population of anglers who consume self-caught fish from the Upper Hudson River. Thus, the exposure and risk analysis assumes the Upper Hudson River accounts for 100% of the sportfish catch of the angler (FS=1). As noted below, the fish ingestion rate is based upon consumption of sportfish, such that it excludes fish that may be purchased and then consumed.

Exposure Frequency (EF). Because the fish ingestion rate is based on an annualized average ingestion over one year, an implicit exposure frequency value of 365 days/year is used in the intake calculation. This does not imply consumption of fish 365 days per year.

Exposure Duration (ED). While Superfund risk assessments typically use the length of time that an individual remains in a single residence as an estimate for exposure duration, such an estimate is not likely to be a good predictor of angling duration, because an individual may move into a nearby residence and continue to fish in the same location, or an individual may chose to stop angling irrespective of the

location of their home. Furthermore, given the large size of the Hudson River PCBs Superfund site, an individual may move from one place of residence to another, and still remain within the Upper Hudson area and continue to fish from the Upper Hudson River. For the purposes of defining the angler population likely to fish the Upper Hudson River most frequently, it was assumed this population would be most likely to constitute residents from the five counties bordering the Upper Hudson River (Albany, Rensselaer, Saratoga, Warren, and Washington). Furthermore, the 1991 New York Angler survey (see Chapter 3 discussion) found that the average distance traveled by New York anglers was 34 miles, supporting the notion that the majority of the angler population for the Upper Hudson River is likely to reside in these counties.

Given the above considerations, the exposure duration (angling, or fishing, duration) for the fish consumption pathway is not based solely upon a typical residence duration. Instead, as described in Section 3.2.4, an angler is assumed to continue fishing until any of the following occur:

- the individual stops fishing;
- the individual moves out of the area, or dies.

The 1991 New York Angler survey of over 1,000 anglers (Connelly *et al.*, 1992) was used to estimate fishing duration habits within the population of New York anglers. U.S. Census data (1990) on county to county mobility provided the source of information to estimate the range of residence durations within the five counties bordering the Upper Hudson River.

The 50^{th} percentile of the fishing duration distribution is 12 years and the 95^{th} percentile is 40 years. These values were used as the central tendency and RME point estimates, respectively. For comparison, 9 years, and 30 years are standard exposure duration factors for Superfund risk assessments based on national statistics of population mobility alone (USEPA, 1989b).

Body Weight (BW). The average adult body weight used in the intake equation was 70 kg, taken from USEPA (1989a). Note that the adult body weight found in the 1997 Exposure Factors Handbook (USEPA, 1997f) is 71.8 kg. Because USEPA's derivation of the PCB cancer toxicity factors was based upon a 70 kg adult in extrapolating the animal data to humans, this assessment uses the prior 70 kg body weight value for consistency (USEPA, 1997b).

Averaging Time (AT). A 70-year lifetime averaging time of 25,550 days was used for cancer calculations (70 years × 365 day/year) (USEPA, 1989a). In order to avoid possible confusion, a 70 year life expectancy from USEPA RAGS was used as the averaging time for cancer, even though the 1997 Exposure Factors Handbook (USEPA, 1997f) indicates 75 years is the most current estimate. Had a 75 year averaging time been used, this would effectively decrease the calculated intake of PCBs in fish by 7%.

Non-cancer averaging times are not averaged over a lifetime, but rather over a period of time equating to a chronic level of exposure. Chronic exposure are those exposures that exceed the subchronic exposure durations (7 years). Because the PCB concentration in fish declines for the projected 70 year period covered by this risk assessment, the average concentration (over time) actually declines as the exposure period increases. Thus, the average concentration (and by extension, average PCB intake in

terms of mg/kg-day) in a 7-year exposure period is actually greater than the average concentration over, say 40 years. This leads to the somewhat counter-intuitive result that the average daily dose decreases as the exposure duration increases. For cancer risk evaluation, which is based upon a lifetime averaging period, this lower average daily dose still yields a higher overall PCB intake, simply because the intake is accumulated over the lifetime. For the evaluation of non-cancer hazards, it is inappropriate to extend the averaging time to equal the exposure duration in this case, because the higher average dose experienced over less than a lifetime of exposure (*e.g.*, 7 years) may exceed an acceptable dose, and may not be representative of an RME exposure.

Based on the foregoing considerations, the averaging time for the non-cancer hazard assessment was set to 2,555 days (7 years × 365 days/year) for the RME point estimate and 4,380 days (12 years × 365 days/ year) for the central tendency estimate.

Concentration of PCB in Fish (C_{fish}). As described earlier in Section 2.3.1, the PCB concentration in fish was determined based on the modeled Total PCB concentration results presented in the Baseline Modeling Report (USEPA, 1999d), combined with the fish consumption patterns as defined by the 1991 New York Angler survey (Connelly *et al.*, 1992). For the evaluation of cancer risks, the central tendency EPC is 4.4 mg/kg PCBs, which was calculated by averaging the species-weighted concentration distribution over the 50th percentile exposure duration estimate (*i.e.*, 12 years). The corresponding RME value is 2.2 mg/kg PCBs, which was calculated by averaging the species-weighted concentration distribution over the 95th percentile exposure duration estimate (*i.e.*, 40 years). It should be noted that the apparent contradiction in EPC, whereby the high-end EPC is lower than the central tendency EPC, is a direct result of the declining PCB concentration in fish over time. Due to this decline over time, the average concentration over the 40-year exposure duration is less than the average concentration over the 12-year period.

As noted above, the averaging time for the non-cancer hazard assessment was limited to a maximum of 7 years for the RME. Thus, the 7-year average EPC in fish for the RME is 5.1 mg/kg PCBs; the central tendency point estimate EPC, which is based on a 12-year exposure duration, is 4.4 mg/kg PCBs.

Fish Ingestion Rate (IR). The fish ingestion rate is based upon an estimate of the long term average consumption of self-caught fish in the angler population, expressed as an annualized daily average rate in units of grams of fish per day (g/day). It is important to note that the ingestion of fish from all sources (*e.g.*, self-caught plus purchased fish) is necessarily greater than or equal to the ingestion rate of only self-caught fish. Because this HHRA examines the risk of PCB intake from Hudson River fish only, the focus is only on self-caught fish.

As described in detail in Section 3.2.1, the fish ingestion rate for the HHRA is based upon a survey of over 1,000 New York anglers (Connelly *et al.*, 1992) who catch and consume fish. For the point estimate exposure and risk calculations, the 50th percentile of the empirical distribution (4.0 g/day) is used as the central tendency point estimate of fish ingestion, and the 90th percentile (31.9 g/day) is the RME ingestion rate.[7] For a one-half pound serving, these ingestion rates represent approximately 6.4 and 51 fish meals per year, respectively.

[7] A fish ingestion rate of 30 grams per day was used in the Phase 1 risk assessment which was the USEPA-recommended value at the time of that report (USEPA, 1991a).

Cooking Loss (LOSS). Numerous studies have examined the loss of PCBs from fish during food preparation and cooking. A review of the available literature is discussed in detail in Section 3.2.3 and a brief summary is presented here.

Experimental results range considerably, both between various cooking methods and within the same method. Cooking losses, expressed as percent loss based on Total PCB mass before and after cooking, as high as 74 percent were reported in one study (Skea *et al.*, 1979). Several studies reported net gains of PCBs (Moya *et al.*, 1998; Armbruster *et al.*, 1987).[8]

Despite a wide range of data covering 12 studies, it is not possible to determine the key factors that influence the extent of PCB cooking losses. PCB losses from cooking may be a function of the cooking method (*i.e.*, baking, frying, broiling, *etc*.), the cooking duration, the temperature during cooking, preparation techniques (*i.e.*, trimmed *vs*. untrimmed, with or without skin), the lipid content of the fish, the fish species, the magnitude of the PCB contamination in the raw fish, the extent to which lipids separated during cooking are consumed, the reporting method, and/or the experimental study design. In addition, personal preferences for various preparation and cooking methods and other related habits (such as consuming pan drippings) may result in consumption of PCBs "lost" from the fish upon cooking.

The 12 studies reviewed (Section 3.2.3) support the conclusion that cooking loss may be zero to 74 percent. Despite the rather wide range of cooking loss estimates, most PCB losses were between 10 and 40 percent. A value of 20% (midpoint of 0% - 40%) was selected as the central tendency point estimate for cooking loss. For the RME, no cooking loss (LOSS = 0%) was selected to include the possibility that pan drippings are consumed.

2.4.2 Ingestion of Sediment

For the sediment ingestion pathway, intake is calculated as:

$$\text{Intake}_{\text{ingestion}}(\text{mg / kg - d}) = \frac{C_{\text{sed}} \times \text{IR} \times \text{FS} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

where:

C_{sed}	=	Concentration of PCBs in sediment (mg/kg)
IR	=	Sediment ingestion rate (mg/day)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

[8] It is likely that the net gain is within the experimental measurement error and essentially indicates zero loss.

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-13 through 2-15. Site-specific considerations in selecting these factors are discussed below.

PCB Concentration in Sediment (C_{sed}). As described in Section 2.3.2, the Baseline Modeling Report (USEPA, 1999d) contains 20-year projections of the PCB concentration in sediment. The mean PCB concentration in sediment of 14.9 mg/kg was used as the central tendency point estimate, and the 95th percentile concentration, 28.7 mg/kg, was used as the RME point estimate.

Sediment Ingestion Rate (IR). This factor provides an estimate of incidental intake of sediment that may occur as a result of hand-to-mouth activity. In the absence of site-specific ingestion rates, USEPA recommended values for daily soil ingestion were used for this factor. The incidental ingestion rate for children is 100 mg/day and for adults and adolescents the value is 50 mg/day. These values, reported as median estimates of soil intake, are the recommendations found in USEPA's current Exposure Factors Handbook (USEPA, 1997f).[9] The incidental soil (sediment) ingestion rate provides an estimate of the ingestion that may occur integrated over a variety of activities, including ingestion of indoor dust. Thus, these median ingestion rates are likely high-end estimates of incidental sediment ingestion while participating in activities along the Hudson, because other sources (such as at home) also account for soil/sediment ingestion.

Exposure Frequency (EF). Exposure to river sediments is most likely to occur during recreational activities. However, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Upper Hudson River, nor are there general population studies that provide usable information. Under the assumption that recreational activities are likely to be most frequent during the summer months, an estimate of one day per week during the 13 weeks of summer is considered a reasonable estimate of the RME value for adults (*i.e.*, 13 days per year). This same frequency was adopted for children (aged 1-6), assuming they would most likely be accompanied by an adult. For adolescents (aged 7-18), who are not as likely to be accompanied by an adult, it was assumed their recreational frequency was three-fold greater than the adult/child frequency (*i.e.*, 39 days per year). The RME values were reduced by 50% for the central tendency exposure calculations. The RME exposure frequency factors used here are approximately 2- to 3-fold higher than the values used in the 1991 Phase 1 risk assessment.

Exposure Duration (ED). The RME exposure duration for sediment ingestion in recreational scenarios is 41 years, and the central tendency value is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the five Upper Hudson counties (see Section 3.2.4.3 and Figure 3-5a). Note the distinction between a RME of 41 years and a central estimate of 11 years for residence duration as opposed to a RME of 40 years and a central estimate of 12 years for angling duration. The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (which sum to 11 years). Note that these values are somewhat greater than values determined from nationwide statistics which indicate 30 years is the 95th percentile and 9 years is the 50th percentile residence duration at one location (USEPA, 1997f).

[9] In the Phase 1 risk assessment, a value of 200 mg/day was used as the sediment ingestion rate for children, and 100 mg/day for adolescents and adults, which were the then recommended high-end ingestion rates prior to the new issue of the 1997 Exposoure Factors Handbook (USEPA, 1997f).

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations (USEPA, 1989a). Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1997f).

2.4.3 Dermal Contact with Sediment

For the sediment dermal contact, absorbed doses are used. Dermal intake (the amount absorbed into the body) is calculated as:

$$\text{Intake}_{\text{dermal}}(\text{mg/kg-d}) = \frac{C_{sed} \times DA \times AF \times SA \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{sed}	=	Concentration PCBs in sediment (mg/kg),
DA	=	Dermal absorption fraction (unitless),
AF	=	Sediment/skin adherence factor (mg/cm^2),
SA	=	Skin surface area exposed (cm^2/exposure event),
EF	=	Exposure frequency (exposure events/year),
ED	=	Exposure duration (years),
CF	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-13 through 2-15. Site-specific considerations in selecting these factors are discussed below.

PCB Concentration in Sediment (C_{sed}). As described in Section 2.3.3, the Baseline Modeling Report (USEPA, 1999d) contains 20-year projections of the PCB concentration in sediment. The mean sediment concentration of 14.9 mg/kg is the central tendency point estimate, and the 95th percentile upper bound segment average of 28.7 mg/kg is the RME point estimated EPC.

Dermal Absorption Fraction (DA). The dermal absorption fraction represents the amount of a chemical in contact with skin that is absorbed through the skin and into the bloodstream. The dermal absorption rate of 14% used in this HHRA is based on the *in vivo* percutaneous absorption of PCBs from soil by rhesus monkeys (Wester *et al.*, 1993).

Soil/Skin Adherence Factor (AF). The sediment adherence values for the risk assessment were obtained from USEPA's March 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f), which among other studies, relies upon data published by Kissel *et al.* (1998). That study represents a

continuation of dermal adherence studies that provide the basis for the current exposure factors recommended by USEPA in its 1997 Exposure Factors Handbook (USEPA, 1997f).

The data in Kissel *et al.* (1998) include soil/skin adherence factors for a range of activities and individuals (*i.e.*, transplanting of bedding plants, laying of pipe by adults, children's play, *etc*.). For each of these activities, Kissel lists measured dermal adherence (soil loadings) on four body parts (hands, forearms, lower legs, and faces). Area weighted adherence factors for the Kissel, *et al.* (1998) study, and others, are presented in the March 1999 Draft Dermal Risk Assessment Guidance. The area-weighted sediment/skin adherence values for adults and children are determined by summing the soil loading rates of each body part (hands, forearms, lower legs and face) multiplied by their respective surface area, and dividing by the sum of the surface areas. The resulting 50^{th} percentile sediment/skin adherence factor for children is 0.2 mg/cm^2, and 0.3 mg/cm^2 for adults (USEPA, 1999f). These adherence factors are for children playing in wet soil, and adults whose soil loadings were measured for reed gathering activities. These activities, which represent active contact with soil, are appropriate surrogates for activities where Upper Hudson River recreators may contact sediment. The soil adherence factor for adolescents was taken as the midpoint between the child and adult factors.

Skin Surface Area Exposed (SA). For children and adolescents, the mean surface area of hands, forearms, lower legs, feet, and face were calculated by multiplying the total body surface area (averaged between males and females) by the percentage of total body surface area that make up the relevant body parts (USEPA, 1997f). For children, the mean surface area of the hands, forearms, lower legs, feet, and face is 2,792 cm^2 (using data for the category 6<7 years); for adolescents, the mean surface area of the hands, forearms, lower legs, feet, and face is 4,263 cm^2 (for age 12 years); the mean surface area of adult hands, forearms, lower legs, feet, and face is 6,073 cm^2 (USEPA, 1997f). In the Phase 1 risk assessment, the corresponding exposure factors used were: 3,931 cm^2, 7,420 cm^2, and 5,170 cm^2 for child, adolescent, and adult surface areas, respectively. These prior values were based upon the surface area of the child/adolescent legs, feet, arms, and hands, and adult lower legs and feet, forearms, and hands.

Exposure Frequency (EF). As described above, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Upper Hudson River, nor do general population studies exist that provide usable information. The exposure frequency factors (Tables 2-13 through 2-15) for dermal contact are the same as those for incidental ingestion described in the proceeding section.

Exposure Duration (ED). The exposure duration for sediment dermal contact in recreational scenarios is 41 years, and the central tendency value is 11 years, which correspond to the 95^{th} and 50^{th} percentiles, respectively, of the residence duration determined for the five Upper Hudson counties (see Section 3.2.4.3 and Figure 3-5a). Note the distinction between a RME of 41 years and a central tendency of 11 years for residence duration as opposed to a RME of 40 years and a central tendency of 12 years for angling duration. The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (which sum to 11 years). Note that these values are somewhat greater than values determined from nationwide statistics which indicate 30 years is the 95^{th} percentile and 9 years is the 50^{th} percentile residence duration at one location (USEPA, 1997f).

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations (USEPA, 1989a). Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1997f).

2.4.4 Dermal Contact with River Water

For the river water dermal contact pathway, dermal intake (the amount absorbed into the body) is calculated as:

$$\text{Intake}_{\text{water}}(\text{mg/kg-d}) = \frac{C_w \times K_p \times SA \times DE \times EF \times ED \times CF}{BW \times AT}$$

where:

C_w	=	Concentration of PCBs in water (mg/l)
K_p	=	Chemical-specific dermal permeability constant (cm/hr)
SA	=	Skin surface area exposed (cm^2)
DE	=	Duration of event (hr/d)
EF	=	Exposure frequency (d/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-3} L/cm^3)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-16 through 2-18. Site-specific considerations in selecting these factors are discussed below.

PCB Concentrations in River Water (C_w). As described in Section 2.3.4, the Baseline Modeling Report (USEPA, 1999d) contains 20-year projections of the PCB concentration in sediment. The mean water column PCB (2.4×10^{-5} mg/L) is the central tendency point estimate EPC, and the 95th percentile upper bound segment average water column PCB concentration (3.1×10^{-5}) is the RME point estimate.

Permeability Constant (K_p). In the absence of experimental measurements for the dermal permeability constant for PCBs, it was estimated to be 0.48 cm/hr based on the value for hexachlorobiphenyls reported in the 1999 Draft Dermal Risk Assessment Guidance (USEPA, 1999f).

Skin Surface Area Exposed (SA). As a conservative estimate of possible exposure, 100% of the full-body surface area was assumed to come into contact with water. The surface areas for adults, adolescents, and children, respectively are: 18,150 cm^2, 13,100 cm^2, and 6,880 cm^2 (USEPA, 1997f).

Duration of Event (DE). For all recreator scenarios, 2.6 hours/day was used as the river water dermal exposure time, which is the national average duration for a swimming event (USEPA, 1989b).

Exposure Frequency (EF). As described above, there are no site-specific data to provide an indication of the likely frequency of recreational activities along the Upper Hudson River, nor do general population studies exist that provide usable information. The exposure frequency factors (Tables 2-16 through 2-18) for dermal contact with water while swimming are the same as those for incidental ingestion and dermal contact with sediments described in the proceeding sections.

Exposure Duration (ED). The exposure duration for river water dermal contact in recreational scenarios is 41 years, and the central tendency value is 11 years, which correspond to the 95^{th} and 50^{th} percentiles, respectively, of the residence duration determined for the five Upper Hudson counties (see Section 3.2.4.3 and Figure 3-5a). Note the distinction between a RME of 41 years and a central tendency of 11 years for residence duration as opposed to a RME of 40 years and a central tendency of 12 years for angling duration. The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (which sum to 11 years). Note that these values are somewhat greater than values determined from nationwide statistics, which indicate 30 years is the 95^{th} percentile and 9 years is the 50^{th} percentile residence duration at one location (USEPA, 1997f).

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a).

Averaging Time (AT). For all recreational exposure calculations, a 70-year lifetime averaging time of 25,550 days (365 days × 70 years) was used for cancer evaluations (USEPA, 1989a). Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1997f).

2.4.5 Inhalation of PCBs in Air

For the inhalation pathway, intake is calculated as:

$$\text{Intake}_{\text{inhalation}}(\text{mg / kg - d}) = \frac{C_{air} \times IR \times DE \times EF \times ED \times CF}{BW \times AT}$$

where:

C_{air}	=	Concentration of the chemical in air ($\mu g/m^3$),
IR	=	Inhalation rate (m^3/hr)
DE	=	Duration of event (hrs/day)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (yrs)
CF	=	Conversion factor (10^{-3} mg/μg)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Exposure factor values for the central tendency and RME point estimate calculations for this pathway are summarized in Tables 2-19 through 2-24. Site-specific considerations in selecting these factors are discussed below.

PCB Concentrations in Air (C_{air}). The exposure point concentration estimates, summarized in Section 2.3.4, were estimated for areas in the immediate proximity of the river. The central tendency point estimate is 1×10^{-6} mg/m^3, the RME estimate is 1.7×10^{-5} mg/m^3.

Inhalation Rate (IR). For adult residents, the inhalation rate used is 20 m^3/day, which is the recommended value for long term exposure assessments for Superfund risk assessments (USEPA, 1991b). The inhalation rate for children (10 m^3/day) and adolescents (13.5 m^3/day) used to calculate inhalation are current recommendations in the 1997 Exposure Factors Handbook for long term exposures (USEPA, 1997f).[10] The same values were used in both central estimate and high-end exposure calculations.

For all recreational scenarios, the mean inhalation rate values for short-term, moderate activities were used: 1.6 m^3/hr for adults and adolescents, and 1.2 m^3/hr for children (USEPA, 1997f).

Exposure Frequency (EF). Because residents may be exposed to PCB-affected air when performing activities outside their homes as well as when they are inside (through outside air exchange), a high-end scenario assuming exposure 24 hours a day, 350 days a year was adopted. The exposure frequency for inhalation of air during recreational activities is the same as those for incidental ingestion of sediment and dermal contact with sediment and river water.

[10] These values are based on children aged 6-8 years and the average male/female adolescent 12-14 year age category.

Exposure Duration (ED). The exposure duration for the inhalation pathway is 41 years and the central tendency value is 11 years, which correspond to the 95th and 50th percentiles, respectively, of the residence duration determined for the five Upper Hudson counties (see Section 3.2.4.3 and Figure 3-5a). Note the distinction between a RME of 41 years and a central tendency of 11 years for residence duration as opposed to a RME of 40 years and a central tendency of 12 years for angling duration. The RME exposure duration is 6 years for children, 12 years for adolescents, and 23 years for adults (summing to 41 years), and the central tendency exposure duration is 3 years for children, 3 years for adolescents, and 5 years for adults (which sum to 11 years). Note that these values are somewhat greater than values determined from nationwide statistics, which indicate 30 years is the 95th percentile and 9 years is the 50th percentile residence duration at one location (USEPA, 1997f).

Body Weight (BW). Age-specific body weights were used. The mean body weight for children aged 1 to 6 is 15 kg, the mean body weight for adolescents aged 7-18 is 43 kg, and the mean adult body weight is 70 kg (USEPA, 1989a).

Averaging Time (AT). A 70-year averaging time of 25,550 days was used for cancer evaluations (365 days/year × 70 years) (USEPA, 1997). Non-cancer averaging times are equal to the exposure duration multiplied by 365 days/year (USEPA, 1997f).

3 Monte Carlo Exposure Analysis of Fish Ingestion Pathway

A Monte Carlo analysis was conducted pursuant to the Agency's guidance on this subject (USEPA, 1997a). The purpose of the Monte Carlo analysis is to estimate a probability distribution of PCB exposure among members of the angler population and to quantify the extent to which some sources of uncertainty affect the precision of these estimates. When combined with the toxicity information described in Chapter 4, the range of PCB exposure is translated into a range of cancer risks and non-cancer hazards (Chapter 5).

As described earlier, USEPA's guidance for Superfund risk assessments and USEPA policy recommends an evaluation of reasonable maximum exposure. In the preceding section, one method of estimating the RME was outlined. The point estimate method consists of combining high-end and appropriate average exposure estimates for exposure factors such that the combination of factors yields an estimate of an individual who may experience a reasonable maximum exposure. While the RME is widely used to capture exposures in the high-end of the distribution (above the 90^{th} percentile), in practice it is rare that the precise probability associated with the RME can be determined. That is, the result is clearly a "high-end" estimate of exposure, but it is difficult to determine whether the high-end is the 75^{th} percentile, 90^{th} percentile, 99^{th} percentile, *etc*. within a population.

Monte Carlo simulation methods provide an alternative, probabilistic, approach to estimate the RME. The advantage afforded by Monte Carlo methods is that, given sufficient data on parameter distributions, they can provide an explicit estimate of the likelihood, or probability, associated with the entire range of exposure -- this quantitative estimate of the probability of exposure translates into a quantitative estimate of the probability of risk as discussed in Chapter 5. The advantages offered by Monte Carlo exposure analysis involve more resource intensive analysis, as well as more detailed information describing the distribution of plausible values for the exposure factors.

After the exposure factor distributions have been determined, performing the Monte Carlo simulation is straightforward: the range and relative likelihood of exposure is calculated by replacing exposure factor point estimate values with values sampled from their respective probability distributions. The simulation randomly selects a value from each parameter's distribution and calculates the corresponding PCB intake, repeating this process many times. The collection of computed PCB intake values approximates the exposure distribution for the population of interest.

Although the actual simulation process is straightforward, the significant challenge of a Monte Carlo analysis lies in developing the probability distributions that describe each exposure factor. The majority of the discussion in this section examines the information sources used to derive the distributions for each of the exposure factors. Furthermore, the uncertainties involved in deriving the input probability distributions are clearly outlined. Before proceeding, the next section highlights the distinction between two important concepts in the analysis, variability and uncertainty, each of which contribute to variations in the exposure calculations.

3.1 Discussion of Variability and Uncertainty

It is important to segregate the influence of variability and uncertainty in the context of the Monte Carlo Analysis because they give rise to two sets of questions. Variability addresses the issue of whether there are members of the population with a particularly elevated level of intake (and by extension risk), whereas uncertainty affects the precision of the intake estimates.

Exposure factors can vary among the population, and they can be uncertain due to limited amount of information. Parameter variability is an inherent reflection of the natural variation within a population (*e.g.*, true differences in fish ingestion rates, exposure duration, body weight, *etc.*). Uncertainty represents a lack of perfect knowledge about specific variables, models, or other factors. Uncertainty can be reduced through further study, measurements, *etc.*, whereas variability cannot. Further study of the variability of the characteristics affecting exposure within a population can however improve the accuracy with which the variability can be modeled and thus can improve the accuracy of exposure and risk estimates.

The exposure factor parameters used to estimate chemical intake, in concept, have multiple possible values for any of three reasons. First, a parameter's true value may be uncertain, but may not vary substantially across different members of the population. In this case, the parameter has one "true" value for all members of the population of interest, but that value is not known precisely. Second, a parameter's value may vary from member to member of the population, but be treated as known with relative certainty. For example, the distribution of human body weights within a population clearly varies, yet given a sufficient number of measurements the variability may be determined with accuracy. Third, a quantity may both be uncertain and vary from member to member of the population. In practice, most exposure factors fall into this third category. Assessments need to address both variability in a population and scientific uncertainty in the risk estimates. The effects of these factors need to be addressed separately and not mixed together in an assessment to develop a single risk distribution. There are different alternatives for presenting information on variability and uncertainty, depending on the available data and assessment needs.

If the distinction between uncertainty of an exposure factor and true variability among the population were not distinguishable, then a single probability distribution for each exposure factor would be all that is needed for a Monte Carlo analysis. In this instance, a "one-dimensional" Monte Carlo analysis would proceed repeatedly drawing randomly selected values for each stochastic parameter (*i.e.*, a random sample reflecting a combination of uncertainty and variability). For each set of values drawn, the simulation computes an intake, repeating this process a large number of times. The resulting set of intake (exposure) estimates can be plotted as a histogram that approximates the range and relative likelihood of the plausible exposure that may exist in the modeled population. However, this approximation to the probability distribution of exposure (and risks) generated by a one-dimensional Monte Carlo simulation has embedded within it both variability and uncertainty. Because it reflects both uncertainty and variability, it is broader than the true distribution of risks. Moreover, it cannot be thought of as representing the risk that any one individual would incur.

A two-dimensional (2-D), or nested Monte Carlo simulation addresses this problem by conducting a large number of separate one-dimensional (1-D) simulations. For each 1-D simulation, a fixed set of randomly selected values is assigned to each of the uncertain parameters. Values for variable parameters

are permitted to vary within each 1-D simulation. Each 1-D simulation produces a large number of intake estimates (*e.g.*, 1,000 to 10,000 or more such estimates) representing the set of PCB intake incurred by members of a population, given the fixed values assigned to each uncertain parameter for that simulation.

The results of a two-dimensional analysis can be used to quantify the distribution of plausible risks for representative members of the population. For example, the range of plausible risks for the "median individual" (*i.e.*, the individual whose risk is greater than the risk for one-half of the population, and less than the risk for the other half) is estimated by collecting the median risk value from each of the 10,000 executed 1-D simulations.

In the Scope of Work of the Phase 2 HHRA (USEPA, 1998a), a 2-D Monte Carlo analysis had been proposed in order to explicitly address uncertainty and variability. The 2-D analysis involves: (1) defining probability distributions that reflect the parameter variability (*i.e.*, true differences in fish ingestion, exposure frequency, exposure duration, body weight, *etc*. within an exposed population), and (2) evaluating the uncertainty associated with the exposure factor distributions. Thus, the first component (variability analysis) of this process yields a probability distribution that conveys information on the range of risk experienced by individuals within a population, and allows a quantitative estimate of the RME individual (such as the 95^{th} percentile exposure and risk). The second component (uncertainty analysis) is intended to provide quantitative estimates of the accuracy of the predictions. Uncertainty in the exposure parameter estimates affects the precision of the resulting risk estimates. The more reliable the information is to define the exposure factor probability distributions, the narrower the range of Monte Carlo exposure estimates for any particular exposure percentile; conversely, greater uncertainty in the exposure factor distributions leads to wider range in the risk estimates.

While a nested Monte Carlo provides a framework for evaluating both the variability of exposure within a population and provides a quantitative estimate of the accuracy of the exposure, the information required to conduct the analysis is substantial. Modeling variability and uncertainty separately requires not only a probability distribution defining the variability for a particular parameter, but also a quantitative measure of the uncertainty for that probability distribution. As an example, consider modeling the variability of a particular exposure parameter, such as fish ingestion, as a lognormal random variable with parameters μ and σ. In order to accomplish a fully 2-D analysis, quantitative uncertainty distributions for both the mean and variance would in theory be necessary, or in other words not only is a probability distribution of fish ingestion required, so too is the probability distribution for plausible values of μ and σ. Clearly such an approach requires much more information than a 1-D analysis, where uncertainty and variability are not distinguished from one another.

For the reasons described later in this section, an explicit 2-D analysis was not performed due to insufficient information available to define quantitative uncertainty distributions for several important exposure factors. The analysis conducted here includes a 1-D Monte Carlo analysis of the *variability* of exposure as a function of the variability of individual exposure factors. The second component of the analysis includes an uncertainty/sensitivity analysis for the important exposure variables. This sensitivity analysis examines changes in the predicted bottom line distribution of population variability when alternative assumptions are made for the distribution of assessment variables. A total of 72 separate combinations of the variable input parameters were examined in the uncertainty analysis. Thus, the likely precision of each percentile of the exposure estimate distribution is not characterized by a specific

probability, but rather the range of exposure estimates for each percentile is presented to give the reader an estimate for how wide or narrow the exposure estimates range.

Before proceeding with the Monte Carlo exposure analysis, it must be noted that as a matter of USEPA policy, the variability and/or uncertainty associated with chemical toxicity is not included quantitatively in a Monte Carlo risk analysis. USEPA recognizes the uncertainty inherent in the determination of cancer and non-cancer toxicity factors, and the uncertainty is factored into the determination of the toxicity factors when they are published in USEPA's Integrated Risk Information System (IRIS). A discussion of the toxicity factor uncertainty is presented in Chapter 4, and in the discussion of uncertainties in Chapter 5.

3.2 Derivation of Exposure Factor Distributions

The Monte Carlo analysis calculates chemical intake *via* fish ingestion based upon the basic intake equation defined in Section 2.3.1, which is repeated here for ease of reference:

$$Intake_{fish}(mg/kg\text{-}d) = \frac{C_{fish} \times IR \times (1 - LOSS) \times FS \times EF \times ED}{BW \times AT} \times CF$$

where:

C_{fish}	=	Species weighted concentration of PCBs in fish (mg/kg)
IR	=	Annualized fish ingestion rate (g/day)
LOSS	=	Cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year),
ED	=	Exposure duration (years),
CF	=	Conversion Factor (10^{-3} kg/g)
BW	=	Body weight (kg),
AT	=	Averaging time (days),

For the point estimate exposure analysis, several parameters (C_{fish} and IR in particular) were based on weighted average inputs based upon species ingestion rates. The Monte Carlo analysis does not adopt weighted averages for these exposure factors. Consequently, the calculation of PCB intake from fish ingestion for the Monte Carlo simulation is the summation of the annualized intake over the exposure duration and over all fish species:

$$Intake = \sum_{f} \sum_{y=1999}^{1999+ED-1} \left(\frac{C_{f,y} \times IR_a \times PCT_f \times (1 - LOSS) \times FS \times EF}{BW_a \times AT} \right) \times CF \qquad [3\text{-}1]$$

where:

Intake	=	PCB intake from all fish species over the exposure duration (mg/kg-day)
$C_{f,y}$	=	PCB concentration in fish species *f* in year *y* (mg/kg)
IR_a	=	Fish ingestion rate (g/day) at age *a* (*a = y - year of birth*)
PCT_f	=	Fraction of annual fish ingestion for species *f* (unitless fraction)
LOSS	=	PCB cooking loss (g/g)
FS	=	Fraction from source (unitless fraction)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (10^{-3} kg/g)
BW_a	=	Body weight (kg) at age *a* (*a = y - year* of birth)
AT	=	Averaging time (days) 70 years × 365 days/yr cancer ED × 365 days/yr non-cancer

In this form of the intake equation, exposure duration (ED), referred to here as the incremental exposure duration, is the number of years until the individual stops fishing in the Upper Hudson River because the angler stops fishing altogether or the angler moves out of the region (or dies). The total dose over the exposure duration is given by summing over the three modeled fish species consumed (denoted by subscript *f*).

The variables in the above equation for which probability distributions or sensitivity analysis ranges were developed include:

IR_a	ingestion rate
$C_{f,y}$	concentration of PCBs in fish
PCT_f	percent of species *f* consumed
LOSS	cooking loss
ED	exposure duration (*e.g.*, fishing duration)
BW_a	body weight

Parameters that were treated as constants in the Monte Carlo analysis, set to the same values as they were in the point estimate analysis, were the following:

FS	Fraction from source (100%)
EF	Exposure frequency
AT	Averaging time

A discussion of the derivation of the variable exposure factors is presented in the following subsections.

3.2.1 Fish Ingestion Rate

The fish ingestion rate term represents the amount of fish an individual consumes on average within the year, annualized such that it is expressed in units of grams of fish per day (g/day). For the HHRA, Upper Hudson River anglers are defined as all individuals who would consume self-caught fish from the Upper Hudson River at least once per year in the absence of fish consumption advisories. The population in question therefore includes a range of infrequent to frequent anglers, who may fish for sport (recreational) or for sustenance (food source).

Based on a review of the available literature and consideration of a number of scientific issues relevant to fish ingestion rates, a probability distribution of fish consumption rates was determined using data from the 1991 New York Angler survey (Connelly *et al.*, 1992) to represent Upper Hudson River anglers. The statistics and percentiles for this distribution are summarized in Table 3-1. The point estimate exposure calculations used the 50^{th} percentile of the distribution (4.0 g/day) and the 90^{th} percentile (31.9 g/day) ingestion rates, corresponding to approximately 6.4 and 51 one-half pound meals per year, respectively. The entire distribution of fish ingestion rates was used in the Monte Carlo analysis to represent variability of fish consumption among the angler population. A discussion of the fish ingestion surveys reviewed, and the derivation of the ingestion rate distribution selected, is presented in the following sections.

3.2.1.1 Summary of Fish Ingestion Rate Literature.

Self-caught fish ingestion rates can vary based on many factors, including: the type of water body (flowing *vs.* still, freshwater *vs.* saltwater), the available fish species, the type of consumer (commercial *vs.* recreational), the preference for specific species, the impact of fishing advisories, weather, and the distance of the angler from the water body (reviewed in USEPA, 1997f). Numerous scientific studies of various water bodies (lakes, rivers, streams, *etc.*) have been conducted to identify fishing patterns (frequency, fishing practices, fish species preference, *etc.*) and fish consumption rates. Because the Upper Hudson River is a flowing body of water, the review of fish ingestion literature focused on studies of anglers fishing in inland flowing waterbodies, also emphasizing studies conducted in the Northeast.

Fish ingestion studies can be either "creel" surveys, where anglers are interviewed in person while fishing, or mail surveys, where anglers (often identified as individuals with fishing licenses) are sent questionnaires in the mail (reviewed in USEPA, 1992d). Creel surveys typically involve interviews with anglers at the dockside requesting information about the fishing activities (fish preference, consumption rates, cooking methods, age, gender, frequency of fishing the specific water body, *etc.*). This survey method can provide information on both licensed and unlicensed anglers, depending upon who is interviewed. Mail surveys typically involve sending questionnaires to licensed anglers requesting information on fishing practices, preferred rivers, lakes or streams, fish consumption, and other information. However, if mailing addresses are obtained from list of licensed anglers, unlicensed anglers will not be represented. A third type of survey, diary surveys, where participants are asked to record the frequency of fish ingestion, the types of fish eaten, and the meal size, require more effort on the part of the survey participants, but are generally assumed to yield more accurate results because the potential recall bias found in the other survey methods is minimized.

1988 New York Angler Survey (Connelly et al., 1990). In 1989, researchers at Cornell University performed a statewide mail survey to determine New York anglers' fishing experiences during 1988 (Connelly *et al.*, 1990). Over 10,000 licensed anglers returned completed surveys regarding fishing preferences and interests. A subset of 200 individuals who did not respond to the mail survey was contacted by telephone to account for potential non-response bias. An estimated 26,870 anglers fished in the Hudson River in 1988. The mean distance traveled by anglers fishing in the Hudson was 34 miles. The mean number of fishing trips per Hudson angler was 8.6 trips, and the mean trip duration was 1.2 days. For all New York anglers, the mean age at which they began fishing regularly was 13.3 years of age. Although anglers were asked to estimate their total annual consumption of fish (fresh or saltwater, sport-caught or purchased), they were not specifically asked about the quantity of self-caught freshwater fish consumed.

1991 New York Angler Survey (Connelly et al., 1992). In 1991, researchers at Cornell performed another statewide mail survey to determine New York anglers' awareness and knowledge of fishing advisories, and to determine fish consumption patterns during the 1991 fishing season (Connelly *et al.*, 1992). A total of 1,030 licensed anglers returned completed surveys. A subset of 100 individuals who did not respond to the mail survey was contacted by telephone to account for potential non-response bias. Anglers were also asked to report the number of fish caught and consumed in 1991 according to fish species and fishing location. The overall mean ingestion rate for New York anglers was 11 sport-caught fish meals in 1991. Analysis of the raw survey data also allowed determination of fish ingestion rates for specific locations or for categories of fishing locations (*i.e.*, rivers *vs.* lakes). About 85% of New York anglers were aware of health advisories for fish, and almost half reported that they would eat more sport-caught fish if there were no problems with contaminants. Most New York anglers reported starting fishing at an early age; the mean age at which anglers began fishing was 14 years of age.

1992 Lake Ontario Diary Study (Connelly et al., 1996). Researchers at Cornell performed a 12-month diary study targeting Lake Ontario anglers fishing in 1992 (Connelly *et al.*, 1996). The goal of the study was to provide accurate estimates of fish consumption among Lake Ontario anglers and to evaluate the effect of Lake Ontario health advisory recommendations. Participants were asked to record all fish consumption and fishing trips for an entire year (1992). Participation was encouraged even if anglers intended to fish infrequently to reduce bias toward only avid anglers. Participants were also contacted by telephone to follow-up every three months. A total of 1,202 anglers agreed to participate initially, but only 516 completed their diary for the entire year. Adjustments were made to account for those with less than a full year participation to address potential biases. In January, 1992, participants were also asked to complete a questionnaire asking for 12-month recall of their 1991 fish consumption, which allowed for comparison of results from mail (recall) surveys and diary studies.

Based on the diary results, average daily consumption of sport-caught fish from all sport sources for Lake Ontario anglers was 2.2 g/day for the 50^{th} percentile, and 17.9 g/day for the 95^{th} percentile (Connelly *et al.*, 1996). For fish from all sources (sport-caught and purchased fish), the average daily consumption for Lake Ontario anglers was 14.1 g/day for the 50^{th} percentile, and 42.3 g/day for the 95^{th} percentile. The overall average sport-caught meal size was 232 g/meal, or approximately one-half pound. The 1991 12-month recall mail questionnaires yielded higher fish ingestion rates than those resulting from the diary data, suggesting that recall bias results in overestimates of fish ingestion (Connelly *et al.*, 1996; Connelly and Brown, 1995). Over 95% of the participants were aware of the New York State health advisory, and 32% indicated that they would eat more fish if there were no health advisories.

Additional Connelly Surveys (Connelly and Knuth, 1993; Connelly et al., 1993). In 1993, researchers at Cornell published two studies – one which evaluated angler knowledge and response to Great Lakes health advisories and assessed communication techniques (Connelly and Knuth, 1993), and one which evaluated health advisory awareness and associated behaviors among Lake Ontario anglers (Connelly *et al.*, 1993). Both reports focused specifically on Great Lakes anglers.

1996 and 1991-1992 Hudson Angler Surveys (NYSDOH, 1999; Barclay, 1993). The New York State Department of Health conducted a creel survey of Hudson River anglers in 1996 (NYSDOH, 1999). This survey used a slightly modified version of the questionnaire and interviewing technique used in a 1991-1992 creel survey of Hudson River anglers conducted by the Hudson River Sloop Clearwater organization (Barclay, 1993). A total of 460 Hudson River anglers were interviewed in the two surveys combined; of these, 132 anglers were from the area between Hudson Falls and the Federal Dam at Troy (the Upper Hudson River). For the following discussion, the 1991-1992 and 1996 surveys are combined and considered a single survey.

Of the Upper Hudson River anglers, over 85% were male; almost all (97%) were Caucasian. About 17% of the anglers were under 20, and almost 10% were 60 and older. Half of those surveyed had a New York fishing license, 8% did not have a license, and 42% did not respond. All of the anglers interviewed from the Upper Hudson River were fishing from shore, and not from a boat. About half of the anglers in the Upper Hudson River area had caught any fish at the time of the interview; the most commonly reported fish caught included smallmouth bass, largemouth bass, and white perch. Blue crabs were caught only south of Catskill, not in the Upper Hudson River (NYSDOH, 1999).

About two-thirds of the Upper Hudson River anglers were aware of official health warnings about eating fish from the Hudson. Only one angler reported food as a main reason for fishing; most anglers were fishing primarily for recreation or other similar reasons. About 92% reported that they never eat their catch, and similarly about 90% reported never giving their catch away to others. Only about 14% of Upper Hudson River anglers reported having eaten fish from the Hudson in the past; of those, about 37% reported eating fish once per week, about 19% reported eating fish 2-3 times per month, another 19% reported eating fish once per month, and 25% reported eating fish less than once per month (NYSDOH, 1999).

About two thirds of the Upper Hudson River anglers reported fishing two times or less in the previous week; six percent reported fishing 7 times in the previous week. On a monthly basis, about half reported fishing three times or less in the previous month; about 12% reported fishing 20 or more times in the previous month. Anglers were not asked about their total number of fishing trips per year (NYSDOH, 1999).

1993 Maine Angler Survey (Ebert et al., 1993). Ebert and colleagues conducted a mail survey of licensed Maine anglers. A total of 1,612 licensed anglers returned completed surveys. Anglers were questioned about the number of fish caught and consumed from flowing and standing water bodies and the number of fishing trips completed in the 1990 season. The study authors developed a distribution of fish ingestion rates assuming that all freshwater fish caught by the angler is shared equally with other household members, with the 50^{th} percentile (median) fish consumption from flowing waters equaling 0.99 g/day, and the 95^{th} percentile equaling 12 g/day. Assuming that only the angler consumes fish and there is

no sharing in the household yielded a distribution with the 50th percentile (median) fish consumption from flowing waters equaling 2.5 g/day, and the 95th percentile equaling 27 g/day.

1990 Mid-Hudson Angler Survey (Jackson, 1990). A survey of Hudson River anglers fishing between Stuyvesant and Kingston (within the mid-Hudson) was conducted by researchers at Cornell University in 1990 (Jackson, 1990). From May to August, 1990, they interviewed 413 individuals fishing from shore and 265 individuals fishing from boats to determine fish species preferences, the percentage of anglers that keep and eat Hudson fish, awareness of fish advisories, and various other characteristics. Over half (57.1%) of the anglers were fishing for "anything", 28.6% were fishing for large or small mouth bass, and 9.3% were fishing for striped bass. Of those interviewed, most were male between the ages of 31 and 60 (82% male, 18% female; 8% <16 years, 10.8% 16-20 years, 29.1% 21-30 years, 44.6% 31-60 years, 7.5% >60 years). There were significant differences between shore and boat anglers; shore anglers tended to be younger, more casual anglers (*i.e.*, fishing for anything), while boat anglers tended to be older and fishing for specific targeted species. Tournaments are popular in this stretch of the Hudson; almost three-quarters of the boat anglers were practicing for or participating in a tournament.

1998 Survey of Hudson River Striped Bass Fishery (Peterson, 1998). The recreational striped bass fishery is an important social and economic resource to residents of eastern New York state (Peterson, 1998). Based on creel surveys of boat and shore anglers on the Hudson, and interviews with more than 2,700 Hudson anglers conducted from April through June of 1997, the New York Cooperative Fish and Wildlife Research Unit at Cornell University estimated that the striped bass fishery supported more than 145,842 angler trips in 1997 (Peterson, 1998). They further estimated that 112,757 striped bass were caught, of which 14,163 (12.5%) were harvested (caught and kept). However, because striped bass are predominantly only located downstream of the Federal Dam in Troy (River Mile 154), striped bass will be quantitatively evaluated in more detail in the Mid-Hudson Human Health Risk Assessment.

3.2.1.2 Fish Ingestion Rate Distribution

Selection of the most appropriate data set for determining a distribution of fish ingestion rates for the Upper Hudson River involved consideration of a variety of factors. Ideally, site-specific fish ingestion data would be the preferred source of information. However, the objective of this baseline risk assessment is to evaluate exposures to PCBs in fish *in the absence of Hudson-specific health advisories on fish consumption*. Hudson-specific fish ingestion information can not be collected at the present time while a catch and release advisory for all fish from the Upper Hudson River remains in place. Thus, while the 1996 and 1991-1992 Hudson Angler Surveys provide useful site-specific information, they can not be used to determine fish ingestion rates for the Upper Hudson River because they were conducted while fish advisories recommended eating no fish from the Upper Hudson River; fishing was prohibited in the Upper Hudson River during the 1991-1992 survey.

Therefore, the other fish ingestion studies were reviewed to determine the study most appropriate to serve as a surrogate for the Upper Hudson River. For angler fish ingestion rates, it is important to consider a variety of factors, including the type of waterbody (marine *vs*. freshwater, flowing *vs*. still water, single waterbody *vs*. multiple waterbodies), the climate, fishing regulations, and the availability of desired fish species (reviewed in Ebert *et al.*, 1994). It is also important to consider any potential biases introduced by the survey method. All survey methods involve some uncertainties and potential biases. Long term mail survey may involve uncertainties in individuals ability to recall their behaviors over time.

Diary surveys depend on individuals consistency in recording their behaviors and accuracy of record keeping may decrease with time. Connelly and Brown (1995) have reported results where mail recall estimates exceeded diary survey estimates, particularly for frequent anglers. Creel surveys (interviewing anglers "on location") have the advantage of providing data specific to active users of a resource, but are thus more likely to interview frequent anglers (Price et al, 1996).

The review of available fish ingestion studies were first limited to those focusing on recreational anglers (as opposed to fish consumption of the general population that includes consumption of purchased fish) fishing on waterbodies in the Northeast. As just indicated, the two Hudson-specific studies (NYSDOH, 1999; Barclay, 1993) can not be used because the information was collected while advisories against consumption of all fish from the Upper Hudson River were in place. The 1990 Mid-Hudson angler survey (Jackson, 1990) and the 1998 survey of the Hudson River striped bass fishery (Peterson, 1998) focus on the lower and mid-Hudson areas and are similarly impacted by the fishing advisories, and therefore cannot be used to develop a distribution of fish ingestion rates for the Upper Hudson River (striped bass are uncommon in the Upper Hudson). The 1988 New York Angler Survey (Connelly *et al.*, 1990) did not collect information on ingestion rates of self-caught freshwater fish. The additional Connelly surveys (Connelly and Knuth, 1993; Connelly *et al.*, 1993; Connelly *et al.*, 1996) focused on fish caught in the Great Lakes, and are not the preferred source of information for developing Upper Hudson River fish ingestion rates due to differences in the types of waterbodies and the primary species present.

The two remaining studies, the 1991 New York Angler survey (Connelly *et al.*, 1992) and the 1993 Maine angler survey (Ebert *et al.*, 1993), are both comprehensive mail surveys of licensed anglers. Summary statistics for total fish ingestion rates from flowing waterbodies, as well as a distribution of ingestion rates, were presented by the study authors for the 1993 Maine angler survey. The distribution of fish ingestion rates from the Connelly *et al.* (1992) study was calculated by analyzing the raw survey data from the 1991 New York Angler survey.

The 1991 New York Angler survey was selected as the primary source of information for the Monte Carlo analysis of fish ingestion rates for Upper Hudson River anglers because the climate and characteristics of other New York waterbodies are more likely to be similar to the Upper Hudson River than Maine waterbodies. Because the Maine survey asked respondents only about total fish consumption from all flowing waterbodies, and not from individual waterbodies separately, it is not possible to screen the Maine dataset for more "Hudson-like" rivers and streams. Furthermore, in the 1991 New York survey, survey information was collected from a subset of non-respondents over the phone, allowing for correction of non-response bias. Such information was not collected in the 1993 Maine survey. As discussed in a later section, the Maine angler survey was used for the sensitivity analysis performed for this assessment.

The probability distribution of fish consumption rates used in this analysis was generated using the data from the 1991 New York Angler survey (Connelly *et al.*, 1992). Survey responses reporting consumption of an unknown amount of fish were not included in the derivation of the fish ingestion rate distribution. Total ingestion rates greater than 1,000 meals of fish per year were also excluded from the resulting distribution, as such responses seem implausible given that three meals every day would total 1,095 meals. In addition, only non-zero ingestion rates were included in the analysis (42.7% of the responses indicated they ate none of their fish).

Connelly *et al*. (1992) report fish ingestion in meals of fish eaten. These data were converted to reflect fish ingestion rates in terms of g/day assuming a meal size of one-half pound (227 grams). This assumption is consistent with the finding by Connelly *et al*. (1996) that the overall average sport-caught meal size among Lake Ontario anglers was 232 g/meal, or approximately one-half pound. An assumed half-pound meal size is also consistent with typical assumptions about meal size made by state agencies and the Great Lakes Sport Fish Advisory Task Force (Cunningham *et al.*, 1990; GLSFATF, 1993; NYSDOH, 1999).

The responses indicating consumption of fish from flowing water bodies were used to derive the fish ingestion rate distribution; responses indicating consumption of fish from non-flowing water bodies were not included. Many of the surveys included fish eaten from unknown water bodies. For these responses, the fish ingestion rates for each angler were scaled based on the following:

$$IR_{scaled} = IR_{Flowing} + IR_{Unknown} \times \frac{IR_{Flowing}}{IR_{Flowing} + IR_{Non-Flowing}}$$

A total of 226 responses formed the basis of the ingestion rate distribution for the survey respondents. For the non-respondents, the type of water body was not reported. For this cohort, the ingestion rate was scaled drawing a random scaling factor, based on the equation above, from the distribution of respondent values.

Figure 3-2a provides a probability plot of the respondent results. The x-axis of this plot (z-value) is the number of standard deviations from the central value (median). The y-axis is the natural log of the ingestion rate. Data that are lognormal will fall on a straight line. The median ingestion rate for the respondents is approximately 4.35 grams/day.

The 1991 Connelly survey ingestion rates were also corrected for non-response bias. A total of 100 of the 919 non-respondents were interviewed by telephone. Of these 100 interviews, 55 indicated they consumed at least one or more meals of their catch. Figure 3-2b provides a probability plot of the 55 non-respondent ingestion rates. The median ingestion rate for this group is approximately 3.11 grams per day.

Although both distributions appear to be approximately lognormal, they failed several goodness of fit tests. Because the survey responses were categorical (*i.e.*, discrete number of meals eaten per year), many of the responses that clustered at the low end of the ingestion distribution (those for responses indicating a single meal per year), tended to cause the data to fail the goodness of fit test. The results for respondents and non-respondents were combined and this combined distribution for the entire population was the basis for the ingestion rate probability distribution for the Monte Carlo simulation. Figure 3-2c shows the probability plot for the combined data set. The median ingestion rate for the combined data sets is 4.1 g/day. The entire empirical dataset (281 responses) was used to generate 1,000 random samples (with replacement) for the Monte Carlo analysis (*i.e.*, a fitted lognormal distribution was not adopted). Summary statistics and percentiles for the fish ingestion rates distribution are summarized in Table 3-1.

3.2.1.3 Sensitivity/Uncertainty Analysis of Fish Ingestion Rates

As the foregoing discussion of the many surveys of fish catch and ingestion from multiple locations in the country indicates, fish ingestion rates vary among anglers, and the rates determined from independent surveys differ from one another. As a sensitivity/uncertainty analysis, the Monte Carlo simulations were conducted using the fish ingestion study results from three other surveys. Summary statistics for each of these studies are provided in Table 3-2.

The fish ingestion rates based on the 1991 New York Angler survey are consistent with the range of ingestion rates found in the fish ingestion studies that provide the foundation of the generic ingestion rates recommended by USEPA in its 1997 Exposure Factors Handbook (USEPA, 1997f). The values in the Exposure Factors Handbook are based on fish ingestion studies from several different freshwater locations within the country. This value is also similar to the NYSDOH assumptions concerning fish ingestion. Note also that the 90th percentile (31.9 g/day) value used for the RME point estimate, is similar to the value of 30 g/day that was used in the Phase 1 risk assessment.

In the current USEPA Exposure Factors Handbook (USEPA, 1997f), the recommended fish ingestion rates for recreational freshwater fish consumption are 8 g/day (50th percentile) and 25 g/day (95th) percentile. These values are based on composite information from the following studies:

- 1992 Maine Angler Survey (Ebert *et al.*, 1993)
- 1992 Lake Ontario Diary Study (Connelly *et al.*, 1996)
- 1989 Michigan Sport Angler survey (West *et al.*, 1989)

As the summary in Table 3-2 indicates, the median fish ingestion value from the 1991 New York Angler study (4.0 g/day) is between the Michigan 1989 study result for recreational fish ingestion (10.9 g/day), and the 1992 Lake Ontario study value for sportfish ingestion (2.2 g/day), and the 1993 Maine Angler study value adjusted for angler consumption of self-caught fish (2.5 g/day). The 95th percentile fish ingestion rate based on the 1991 New York Angler survey (63.4 g/day) is greater than the corresponding 95th percentile ingestion rates for the three above studies. The 90th percentile from the 1991 New York Angler Survey (31.9 g/day) appears to be more consistent with the 95th percentiles of the other studies summarized in Table 3-2. Plots of the relative frequency distributions of fish ingestion for the four studies used in the sensitivity/uncertainty analysis are provided in Figures 3-3a through 3-3d. For each of the three additional studies used in this analysis, fish ingestion was modeled as a lognormal variate with distribution parameters summarized on the respective figures.[11]

The central and high-end fish ingestion rates for all flowing waterbodies from the 1993 Maine Angler Survey (Ebert *et al.*, 1993), particularly the results assuming that only the angler consumes sport-caught fish and that fish is not shared in the household, are reasonably consistent with the results for all flowing waterbodies from the 1991 New York Angler survey (Connelly *et al.*, 1992). Compared to the 1992 Lake Ontario diary study (Connelly *et al.*, 1996), the ingestion rates for sport caught fish are also

[11] The distribution parameters for the Connelly *et al.* (1996) and West *et al.* (1989) studies were estimated by the best-fit line through the percentiles reported in the 1997 Exposure Factors Handbook (USEPA, 1997f) fit to a lognormal distribution. The R-squared for these regressions were 0.98 and 0.96, respectively.

reasonably consistent, although the values from the 1991 New York Angler survey are somewhat higher. This may be due to differences between Great Lakes anglers and other New York State anglers, or may reflect the fact that the 1992 Lake Ontario study was based on diary records (believed to be more accurate) while the 1991 New York Angler survey was a mail recall survey (possibly biased high due to recall bias). The difference between the two studies is greater for the 95th percentile values, consistent with the findings of Connelly and Brown (1995) that recall bias tended to result in greater overestimation of fishing activities among more frequent anglers than among less frequent anglers. The 95th percentile fish ingestion rate for flowing waterbodies from the 1991 New York Angler survey (Connelly *et al.*, 1992) is somewhat higher than the 95th percentile fish ingestion rate for Lake Ontario anglers for fish from *all* sources (including sport-caught and store-bought fish). Although the above factors may be suggestive that the rates from the 1991 New York survey may be overestimates, the differences could also be attributable to the different types of water bodies covered by the two surveys, and possible differences in fishing patterns among residents of the two states. The 90th percentile ingestion rate from the 1991 New York Angler Survey (Connelly *et al.*, 1992) was adopted as the RME point estimate.

Comparison to the 1996 and 1991-1992 Hudson angler surveys (NYSDOH, 1999; Barclay, 1993) is more complicated. While these studies focused on anglers fishing along the Hudson River, which is of direct interest for this risk assessment, the fact that a catch and release program is in place and current advisories recommend eating no fish from the Upper Hudson River has likely impacted fish ingestion rates. Very few Upper Hudson River anglers currently eat fish from the Upper Hudson; 92% reported never eating their catch. Only 14% reported eating Hudson fish in the past; of those, 6 respondents reported eating fish once per week, 6 respondents reported eating fish one to three times per month, and 4 respondents reported eating fish less than once per month. However, it is difficult to extrapolate these values to annual average ingestion rates, due to seasonal variations in freshwater fishing. Nonetheless, despite the uncertainties in interpreting the fish ingestion data from the Hudson angler surveys, the distribution of fish ingestion rates from the 1991 New York Angler survey seems reasonable, and appears to span the range of consumption rates reported in the Hudson angler surveys.

3.2.1.4 Discussion of Additional Considerations

Licensed Versus Unlicensed Anglers. The 1991 New York Angler survey, used to generate a distribution to represent fish ingestion rates for the Upper Hudson River, was sent only to licensed anglers; unlicensed anglers were not represented in the survey. It is therefore somewhat uncertain whether unlicensed anglers are adequately represented in this risk assessment. However, given that the distribution of fish ingestion rates from the 1991 New York Angler survey seems to span the range of consumption rates reported in the Hudson angler surveys, which included both licensed and unlicensed anglers (as discussed above), it seems likely that unlicensed anglers are reasonably well represented.

Highly Exposed Subpopulations. Subpopulations of highly exposed or lesser exposed anglers have not been explicitly characterized, but instead are assumed to be adequately represented in the fish ingestion rate distribution used for this assessment. For example, the 99th percentile fish ingestion rate from the 1991 New York Angler survey is 393 meals per year, or over one meal per day (Table 3-1). Furthermore, even those responses up to 1,000 meals per year were included from the New York Angler survey. Although it is possible that there are subsistence or highly exposed individuals who do not obtain fishing licenses, and therefore would not have been captured in the 1991 New York Angler survey or

included in the generated distribution of ingestion rates, there are no known, distinct subpopulations that may be highly exposed (such as a Native American community) in the Upper Hudson River area.

Review of the limited literature available on subsistence or highly exposed angler populations supports the assumption that these subpopulations are likely to be adequately represented in the total distribution of fish ingestion rates developed for Upper Hudson River anglers. As presented in a thesis by Wendt entitled "Low Income Families' Fish Consumption of Freshwater Fish Caught From New York State Waters," low-income families in 12 counties throughout New York, including Albany and Rensselaer counties were interviewed (Wendt, 1986). Wendt reported that between 9% and 49% of the low-income families in each county ate freshwater fish from New York State waters. Wendt then conducted a more in-depth survey of low-income families in Wayne County, New York, bordering Lake Ontario and determined fish consumption rates. The average consumption rate was 17.5 meals per year, or 10.9 g/day. In comparison, the arithmetic average consumption rate from the distribution selected to represent Upper Hudson River anglers is 27.8 meals per year, or 17.3 g/day.

As another surrogate for highly exposed angler populations, fish ingestion rate values for Mohawk women, members of a Native American community along the St. Lawrence River who may be more dependent on local fish and game than other subpopulations, were also considered (Fitzgerald *et al.*, 1995). Fitzgerald *et al.* (1995) report the mean number of local fish meals per year consumed by Mohawk women (one year before a pregnancy) was 27.6 meals per year, which falls between the 80^{th} and 90^{th} percentiles of the distribution of fish ingestion rates developed for Upper Hudson River anglers.

Impact of Advisories. The NYSDOH issues numerous health advisories on eating sportfish from New York State rivers, lakes and streams. It is likely that the fish advisories currently in place throughout New York State, and those in the past, have impacted fish ingestion rates from the 1991 New York Angler survey to some degree. Almost half of the respondents in the 1991 New York Angler survey indicated they would eat more sport-caught fish if there were no contamination problems (Connelly *et al.*, 1992). The general state-wide advisory limits the number of sport-caught fish eaten from New York waters to no more than one meal per week (NYSDOH, 1998; NYSDOH, 1999). Some of these general regulations are not health based, but presumably are established to prevent depletion of fisheries. For the Upper Hudson River, from Hudson Falls to the Troy Dam, there is a specific recommendation to eat no fish. For the Mid and Lower Hudson, there is a specific recommendation that women of child-bearing age and children eat no fish, and advisories recommending restrictions on quantities and species consumed for the remaining population.

However, fish advisories are not 100% effective in preventing or limiting fish consumption. Based on an analysis of the raw survey data from the 1991 New York Angler survey (Connelly *et al.*, 1992), there was no significant difference in the mean number of freshwater fish meals eaten when comparing New York waterbodies with full, partial, or no advisories, despite the expectation that the fishing advisories would likely suppress fish ingestion rates to some degree.

To characterize fish ingestion rates that have not been influenced by the Hudson-specific health advisories to eat no fish, this risk assessment uses fish ingestion rates from all flowing waterbodies from the 1991 New York Angler survey (Connelly *et al.*, 1992). The effect of general, non-specific NYSDEC and NYSDOH fishing regulations that would be in effect regardless of PCB contamination levels in the

Hudson River inherently will be taken into account because these regulations also apply to the New York flowing waterbodies surveyed in the 1991 New York Angler survey.

Women and Children Anglers. Although children and adolescents are not required to have fishing licenses in New York State, several sources indicate that many children consume sport-caught freshwater fish as well as adults (Connelly *et al.*, 1990; Connelly *et al.*, 1992; Wendt, 1986). However, ingestion rates of freshwater fish specific for children are not available. The New York Angler surveys provide data on the age at which anglers begin fishing, and this information has been incorporated into the exposure duration modeling to generate both the length of exposure and also the age at which exposure begins. For each modeled angler whose exposure begins during childhood (as shown in Figure 3-4c, approximately 16% of the anglers in the 1991 New York Angler survey were 10 years old, or younger), the same distribution of number of meals per year generated for adult anglers was used, simply scaled according to body weight, on a year by year basis. Thus, children are represented in this risk assessment to the same extent that they are represented in the New York angler populations. Similarly, although fewer women tend to fish than men, women anglers are represented in this risk assessment to the same extent that they are represented in the New York angler populations.

Recall Bias. The 1991 New York Angler survey (Connelly *et al.*, 1992), as well as the other mail recall surveys, may be subject to recall bias. It is difficult for many individuals to remember accurately their activities over an entire year. When asked about recreation participation over a long period of time (*i.e.*, one year), respondents tend to overestimate their activities (reviewed in Connelly and Brown, 1995; Westat, 1989). With respect to fishing specifically, Connelly and Brown (1995) found that anglers reported significantly higher rates of fish consumption and numbers of days fished in 12-month mail recall surveys compared to 12-month diary studies. The difference was greater for anglers who fished more frequently than those who fished less frequently. These results suggest that the data from the 1991 New York Angler survey (Connelly *et al.*, 1992), used to generate the distribution of fish ingestion rates used in the base case analysis in this risk assessment, are more likely to overestimate, rather than underestimate, actual ingestion rates, particularly for more frequent anglers.

Single Versus Multiple Waterbodies. By deriving the distribution of fish ingestion rates from the data for all flowing waterbodies from the 1991 New York Angler survey, it was conservatively assumed that the amount of fish an individual would consume from the Upper Hudson River, a single waterbody, is equal to the amount of fish consumed by New York anglers from all flowing waterbodies. Although this assumption may overestimate fish ingestion rates for anglers who fish in multiple water bodies (including the Upper Hudson River), many of the respondents in the 1991 New York survey fished in only one or two locations; 35.5% fished in only one location and 21% fished in only two (Connelly *et al.*, 1992). For anglers who fish only the Upper Hudson River, the ingestion rate distribution used here would not necessarily overestimate their fish consumption rate.

3.2.2 PCB Concentration in Fish

As described earlier in Section 2.3.1, there are several important environmental factors that affect the determination of the exposure point concentration in fish (C_{fy}) and therefore influence the variability of PCB intake *via* fish ingestion:

1. The concentration of PCBs in any particular species varies for a particular year, but overall it declines over time.

2. The concentration of PCBs within the same fish species varies depending on the location in the Upper Hudson River (higher concentrations upstream than downstream within the same fish species)

3. The PCB concentration varies among different fish species.

Within Species Annual Variability ($C_{f,y}$)

As was discussed in Section 2.3.1., the variability of model-predicted 50th (median) and 95th percentile PCB concentration within fish for any particular year varies by approximately a factor of 2- to 3-fold. It is unknown to what degree the modeled range represents true variability that is expected among fish of the same species, and to what extent the modeled range is a function of model uncertainty. Regardless of the contribution these two factors may represent, the modest range between the 50th and 95th percentile predictions is not anticipated to yield large differences in the mean PCB concentration in fish that are ingested. This conclusion is supported by an examination of the historical sampling results as well.

Based on the historical monitoring data summarized in the Phase 1 Report (Tables B.3-16 through B.3-18), the coefficient of variation (CV), which is the ratio of the standard deviation divided by the mean, of the measured PCB content in brown bullhead and largemouth bass is generally less than 1.0, and typically around 0.7. Compared to this, the upstream to downstream difference in PCB concentration within a given fish species and year is on the order of 2 to 3-fold. Thus, for an angler who consumes a large amount of fish (*i.e.*, someone at greatest risk), the within-species coefficient of variation is typically less than the variation in concentration attributable to fishing either up- or downstream (*i.e.*, fishing location component of variability). Furthermore, the difference in PCB concentration across fish species is also on the order of 2-fold, again greater than the within species coefficient of variation. Thus, even if the within-species annual variability of PCB concentration in fish were included quantitatively in the Monte Carlo analysis, it would likely be overshadowed by the larger variability in concentration across locations and species.

For the above reasons, the within species PCB concentration for any particular year ($C_{f,y}$) was set to the mean modeled concentration for that species and year for the intake calculated using Equation [3-1]. The variability (randomness) of PCB ingestion from fish was modeled based on the variability in the species consumed, which is accounted for by the PCT_f term in Equation [3-1].

Variability of Species Ingested (PCT_f)

As described in Section 2.3.1, the fish species consumption patterns for the point estimate exposure calculations were based on a weighted average of the species consumed. The species consumption weights were based on the 1991 New York Angler surveys (Connelly *et al.*, 1992) which provided information on the fish species caught and consumed by the surveyed anglers.

For the Monte Carlo analysis, the survey responses from all respondents were used to develop a distribution of fish species ingestion patterns. The same criteria applied to fish ingestion, only those angler responses indicating consumption of at least one and fewer than 1,000 meals from flowing water bodies only, were used to derive the species ingestion distribution. This survey group consists of 226 respondents.

A summary of the species ingestion responses for these respondents is presented in Table 3-3. As described earlier in Section 2.3.1, these species were grouped such that only those responses indicating consumption of fish potentially inhabiting the Upper Hudson River were used. These responses were grouped such that each of the three modeled species provided a surrogate for the concentration of any fish within the group.

The fish species reported consumed by the 226 respondents were grouped into one of three groups according to the groupings given in Table 3-4. For the Monte Carlo analysis, random samples (with replacement) were drawn from this empirical distribution of 226 respondents. This distribution ranges from respondents indicating consumption of a single species, to respondents indicating consumption of multiple species.

3.2.3 Cooking Loss

Numerous studies have documented a loss of PCBs from fish due to cooking (Ambruster *et al.*, 1987; Ambruster *et al.*, 1989; Moya *et al.*, 1998; Puffer and Gossett, 1983; Salama *et al.*, 1998; Schecter *et al.*, 1998; Sherer and Price, 1993; Skea *et al.*, 1979; Smith *et al.*, 1973; Wilson *et al.*, 1998; Zabik *et al.*, 1979; Zabik *et al.*, 1995a; Zabik *et al.*, 1995b; Zabik *et al.*, 1996; Zabik and Zabik, 1996). These studies were reviewed to determine if the extent of PCB losses during cooking have been adequately characterized in the scientific literature to support a quantitative estimate of cooking losses for risk assessment purposes. A summary of the cooking loss estimates for each of these studies is provided in Table 3-4.

As this table shows, experimental results range considerably, both between various cooking methods and within the same method. Most PCB losses (expressed as percent loss based on Total PCB mass before and after cooking) were between 10 and 40 percent. Losses as high as 74 percent were reported in one study (Skea *et al.*, 1979). Net gains of PCBs were reported in several studies (Moya *et al.*, 1998; Armbruster *et al.*, 1987).[12] Overall, these studies support the conclusion that some PCBs are lost during cooking. Consistent with this conclusion, both the NYSDOH and the Great Lakes Sport Fish Advisory Task Force recommend proper methods of trimming, skinning, and cooking fish to remove fat and reduce levels of PCBs and other contaminants (NYSDOH, 1998; NYSDOH, 1999; GLSFATF, 1993).

Although cooking loss appears to occur, the extent of PCB cooking losses has not been well characterized in the published literature, and quantitative estimates of cooking losses remain uncertain. There were no consistent differences in PCB losses between cooking methods in the studies reviewed. Although losses from baking were greater than losses from pan-frying in two studies where the same fish type was used for both cooking methods (Ambruster *et al.*, 1987; Salama *et al.*, 1998), the study by Salama *et al.* only used one fish per cooking method, and is therefore of limited significance. It is difficult

[12] It is likely that the net gain is within the experimental measurement error and essentially indicates zero loss.

to make comparisons between different fish types, as different preparation and cooking methods were often used for different fish types. With regards to preparation technique, while data from Zabik *et al.* (1979) and Salama *et al.* (1998) showed greater losses of PCBs from fish cooked with the skin off as compared to skin on, Zabik *et al.* (1995a) observed minimal differences in PCB losses between fish with skin on or skin off.

Based on the available data, it is not possible to quantify the importance of specific factors influencing the extent of PCB cooking losses. PCB losses from cooking may be a function of the cooking method (*i.e.*, baking, frying, broiling, *etc*.), the cooking duration, the temperature during cooking, preparation techniques (*i.e.*, trimmed *vs.* untrimmed, with or without skin), the lipid content of the fish, the fish species, the magnitude of the PCB contamination in the raw fish, the extent to which lipids separated during cooking are consumed, the reporting method, and/or the experimental study design. The extent of reduction of PCBs due to cooking may also depend on the homologues present in the fish. Zabik *et al.* (1994), as cited in Zabik and Zabik (1996), found that cooking losses of pentachloro-, hexachloro- and heptachlorobiphenyls are greater than losses for homologues with either more or fewer chlorines. Differences among the techniques used for extracting and measuring PCBs are another factor that could contribute to the observed differences in cooking loss between studies.

The wide variation in PCB losses observed, both between and within studies, the lack of an association with various factors which could affect PCB losses, and the fact that personal preferences for various preparation and cooking methods and other related habits (such as consuming pan drippings) are poorly defined, highlights that there are many uncertainties associated with estimating losses of PCBs from fish. It is not possible to develop a probability distribution representing the variability of cooking loss expected either among different consumers, or due to different preparation methods. Thus, for the Monte Carlo analysis, cooking loss was held constant. However, for the sensitivity, or parameter uncertainty analysis, the following range of cooking loss were examined:

RME Exposure:	0%
Central tendency estimate:	20%
Low-end exposure estimate:	40%

Although it is possible that PCBs volatilized during cooking could be inhaled, in the absence of any scientific studies in this area, it is not possible to quantify the potential risks or hazards from this pathway. Based on a qualitative assessment of the cooking frequency for fish, the temperatures used in the cooking, the various cooking practices used, and the relatively low toxicity of inhalation *versus* ingestion of PCB contaminated fish, the risks from inhalation while cooking are unlikely to be significant compared to the ingestion of fish.

3.2.4 Exposure Duration

While Superfund risk assessments typically use the length of time that an individual remains in a single residence as an exposure duration, such an estimate may not be a good predictor of angling duration for this assessment, because an individual may move into a nearby residence and continue to fish in the same location, or an individual may chose to stop angling irrespective of the location of their home.

For the fish consumption pathway, this HHRA defines Exposure Duration (ED) to be the number of years, starting in 1999, that an individual consumes fish from the Upper Hudson River. The angler population has been defined as those individuals who consume self-caught fish from the Hudson at least once per year, in the absence of a fishing ban or health advisories. Although the population of anglers who fish from the Upper Hudson River is likely to include individuals from a large geographic area, it was assumed that individuals residing in any of the five counties bordering the Upper Hudson would be the most frequent anglers (recall the 1988 New York Angler survey reports the mean distance traveled by anglers fishing in the Hudson was 34 miles). For members of this population of anglers, exposure is assumed to continue until any of the following occur:

- The individual stops fishing;
- The individual moves out of the area; or
- The individual dies.

Information regarding the age distribution of New York anglers, including the number of years fished, and when anglers began fishing, was obtained from the 1991 New York Angler survey (Connelly *et al.*, 1992). The probability of moving into and out of any of the five counties bordering the Upper Hudson River was derived from 1990 U.S. census data on county-to-county mobility.

As described in the following subsections, determining the distribution of exposure duration for the angler population involves the following computational steps:

1. *Section 3.2.4.1*. The individual's current age and age at which he or she began fishing is randomly drawn from a distribution developed from information contained in the 1991 New York Angler survey conducted by Connelly *et al.* (1992).

2. *Section 3.2.4.2*. The time remaining until an individual stops fishing, which is a function of current age and the age at which the individual started fishing, is derived from the 1991 New York Angler survey data (Connelly *et al.*, 1992).

3. *Section 3.2.4.3*. The time remaining until that individual moves out of the Upper Hudson counties (one of the five counties comprising the Upper Hudson region) is drawn from a distribution developed from the 1990 U.S. Census In-Migration data tape. This distribution describes the time until an individual moves out of the region as a function of current age.

As was discussed earlier in Section 2.4.1, the 50th percentile exposure duration was determined to be 12 years, and the 95th percentile exposure duration is 40 years. The derivation of the distribution is described below.

3.2.4.1 Joint Distribution for Current Age and Fishing Start Age

The joint distribution for current age and the age at which individuals started fishing (the "fishing start age") were characterized from the 1991 New York Angler survey (Connelly *et al.*, 1992). For each

of the 1,030 survey respondents, the survey lists the current age and the age at which the respondent started fishing. In addition to the 1,030 respondents, there were also 919 nonrespondents, of whom 100 were surveyed by telephone. However, the follow-up survey of the non-respondents did not record the age at which these individuals started fishing.

From the 1991 New York Angler survey, the probability that a randomly selected angler started fishing at age *s* and is currently age *c* is denoted *P(s,c)* can be computed as:

$$P(s,c) = \frac{N(s,c)}{\sum_{i,j} N(i,j)} \qquad [3\text{-}2]$$

where

P(s,c)	=	probability of starting fishing at age *s* for individual who is currently age *c*
N(s,c)	=	number of survey individuals who started fishing at age *s* and are now age *c*

The summation in the denominator of Equation [3-2] is simply the summation over all the anglers in the survey. Before conducting these calculations, two adjustments were made to the data, as described below.

Adjustment 1: Data Sparseness. The data were aggregated into 10-year age groups because the value of *N(s,c)* was often small or 0 for some age groups, thus compromising the robustness of the calculated value, *P(s,c)*. Thus, both *s* and *c* were rounded to the nearest value of 10. This aggregation puts a lower limit of 10 years on the age at which individuals start fishing, and hence a lower limit on the age at which exposure may begin. If younger children fish or consume fish caught by others, this aggregation will underestimate exposure somewhat during childhood.

Adjustment 2: Connelly follow-up survey of non-respondents. The Connelly respondent data (N = 1,030) were adjusted to reflect the non-respondent data (N = 913). As noted in Section 3.2.1.1, Connelly *et al.* (1992) resurveyed 100 of the non-respondents and reports the ages of these individuals. However, the non-respondent survey results do not report the age at which non-respondents started fishing. In order to include the non-respondent information in Equation [3-2], the results for the 1,030 initial respondents were therefore adjusted by multiplying *N(s,c)* in Equation [3-2] by an scaling factor (k_c) computed as:

$$k_c = \frac{\frac{913}{100} \times NR(c) + \sum_{s \in all\ start\ ages} N(s,c)}{\sum_{s \in all\ start\ ages} N(s,c)} \qquad [3\text{-}3]$$

where *NR(c)* is the number of resurveyed non-respondents who report their current age to be *c*. This adjustment is based upon the following assumptions:

- The current age of the entire non-respondent group (913) mirrors the current age of the 100 surveyed non-respondents; the factor 913/100 is simply a weighting factor that conveys this adjustment.

- The distribution of the current age for the non-response group is similar to the distribution of current age for the survey respondents.

Discussion of Assumptions

There are several basic assumptions made in deriving the joint distribution for current age and fishing start age, which are summarized here.

- The angler population is a steady state population, meaning that the age profile of this population remains unchanged over time.

- A corollary to the steady state assumption is that the 1991 New York Angler survey is representative of anglers in 1999.

- Information about the 913 non-respondent group can be inferred from the information gathered from 100 non-respondents who were recontacted by Connelly *et al.* (1992).

- Connelly *et al.* (1992) report the current age for the non-respondents, but not the age at which they started fishing. Therefore, the results from the respondents were stratified by current age as a surrogate. The validity of this approach rests on the assumption that the response rate depends statistically on current age but not the age at which an individual starts fishing.

- Although the 1991 New York Angler survey (Connelly *et al.*, 1992)provided information about the reported age at which each angler started fishing, the analysis required grouping the starting age into 10-year age groups. Thus, all starting ages between 5 and 15 years were categorized in the "10 year" age group. This aggregation required an assumption that no one began fishing before 5 years of age, when in fact, 2.9% of the respondents reported starting fishing before age 5.

The survey results suggest that the assumption that the age profile of the angler population remains constant over time is not strictly true, even after they have been adjusted to reflect the data gathered from the resurveyed non-respondents. Specifically, it appears that the survey under-counted the number of young anglers (age 10). The constructed distribution was adjusted, although it is not clear if the adjustment is sufficient to represent of all young anglers. Although the steady state assumption may not be strictly true, there are no studies that have evaluated fishing populations over time. The cross-sectional design of the Connelly *et al.* (1992) study provides a representative indication of fishing activities in the future and is believed to be a reasonable use of available data.

Upper Hudson River Angler Populations Considered

The HHRA is an evaluation of current and future human exposure (and risks). For the purposes of the exposure calculations, the starting year for this evaluation is 1999. Two populations of anglers were considered in the exposure analysis, because it was unclear *a priori* which group might have a longer possible exposure duration. The two groups considered were:

- *The population of all anglers currently living in the five counties of the Upper Hudson region.* For this population, all data from the 1991 New York Angler survey were used to calculate the joint distribution for current age and fishing start age.

- *The population of anglers living in the five counties who started fishing in 1999*: Analysis of the 1991 New York Angler survey data was restricted to individuals who "recently" started fishing. Ideally, these data would include only those anglers whose start age and current age are exactly the same (*i.e.*, individuals who started fishing within the last year). However, restricting the analysis to these individuals resulted in too small a data set. All anglers whose *rounded* fishing start age and current age were the same were used for this analysis.

After evaluating the data for both possible population groups, it turns out that the exposure duration distributions for these two groups did not differ appreciably. Therefore, the Monte Carlo analysis was based upon the "all angler" category. This category also represents a larger set of the New York Angler survey respondents.

3.2.4.2 Time Remaining Until an Individual Stops Fishing

The time remaining until an individual stops fishing was also based upon the 1991 New York Angler survey (Connelly *et al.*, 1992). Because time until an individual stops fishing was not directly available from the Connelly *et al.* (1992) survey, it was estimated using the start age and current age of the respondents. The probability that an individual whose start age is s and whose current age is $c \geq s$ stops fishing within the next T years, designated $F(s,c,T)$, is

$$F(s,c,T) = \frac{N(s,c) - N(s,c+T)}{N(s,c)} \qquad [3\text{-}4]$$

where as defined in the previous section, $N(s,c)$ is the number of individuals in the survey who started fishing at age s and are now age c.

The reasoning underlying Equation [3-4] is that $N(s,c)$ is the number of individuals in a cohort that started fishing at age s and who are now age c, and $N(s,c+T)$ is the number of individuals remaining in this cohort T years in the future. Since the number of individuals who will remain in this cohort T years in the future is unknown, the number of individuals who started fishing at age s and who are currently $c+T$ years of age serves as a surrogate. This approach presumes that the angler population is in a "steady state," meaning that $N(s,c)$ remains unchanged over time for all values of s and c. From this assumption, it also follows that:

- $F(s,c,T)$ must remain unchanged over time; and
- $N(s,c) \geq N(s,c,T)$ for all positive values of T.

Before making these calculations, three adjustments were made to the data. The first two, to address data sparseness and to incorporate the Connelly *et al.* (1992) follow-up survey of non-respondents, are identical to the adjustments described in Section 3.2.4.1. A third adjustment was made to preserve the assumption of steady state. It turns out that even after adjustment of the Connelly *et al.* (1992) data to reflect non-respondents, the condition $N(s,c) \geq N(s,c,T)$, which follows from the steady state assumption, failed to hold true in some cases. There are several possible reasons for this phenomenon, among which are:

- The steady state assumption is not strictly true, and the number of individuals that started fishing at age *s*, $T+c$ years ago exceeds the number of individuals who started fishing c years ago at age *s*;
- The Connelly *et al.* (1992) survey, even after adjustment for non-respondents, still under counts the number of individuals in some age groups.
- The condition may fail due to the sparseness of data for some age groups (*e.g.*, it could be an artifact of sample size and the necessity to aggregate data).

Although the steady state assumption, may not hold exactly, it is believed to be a reasonable approximation. To adjust the survey data so that they are consistent with the steady state assumption (and in order to make it possible to calculate valid values for $F(s,c,T)$), the adjusted counts of survey respondents ($N_{Adj}(s,c)$) were set equal to the maximum of N(s,c) and N(s,c+10). In cases where this adjustment was necessary, the resulting estimate of $F(s,c,10)$ is 0.

The above adjustment may err on the side of understating the probability that an individual will stop fishing within some time period since the value of $N_{Adj}(s,c)$ may exceed $N(s,c+10)$. On the other hand, in cases where the survey under-reported $N(s,c,T)$ for some relatively small value of T, these calculations will overstate the probability that individuals who started fishing at age s and whose current age is c will soon stop fishing.

Summary of Fishing Cessation Probability

A frequency histogram fishing cessation probability is shown in Figure 3-4a. This figure indicates the relative frequency of those anglers who will stop fishing in the given number of years. Thus, approximately 24% of the angler population is estimated to cease fishing in 10 years, approximately 23% in 20 years, 20% in 30 years, *etc*. Approximately 1% are estimated to cease fishing in 70 years.

Figures 3-4a, 3-4b, 3-4c, and 3-4d summarize the fishing cessation age, starting age, current age, and total fishing duration frequency histograms for the angler population. Note that P(s,c) and F(s,c,T) represent conditional probability functions, and cannot be represented with a single histogram.

3.2.4.3 Determination of Residence Duration

The second determinant of total exposure duration is the residence duration in any of the five Upper Hudson counties. The five counties adjacent to the river north of Troy include Albany, Rensselaer, Saratoga, Warren, and Washington. When an individual moves out of these five counties, regular fishing in the Upper Hudson River is assumed to stop.

The distribution for the time remaining until an individual moves out of the Upper Hudson Region is given by estimating the one-year probability that an individual moves out of the region, and then combining these one-year probabilities to calculate the likelihood that an individual will move out of the area over a more extended time period. Specifically, designate $p_{k,n}$ to be the probability that an individual who is now age k moves out of the area in exactly n years. Then $p_{k,n}$ can be computed from the 1-year move probabilities as

$$p_{k,n} = \left[\prod_{i=1}^{n-1} (1 - p_{k+i-1,1}) \right] \times p_{k+n,1} \qquad [3\text{-}5]$$

where the product (indicated by the $\prod$ symbol) is taken over a series of terms indexed by the subscript i. Note that the product within the brackets is the probability that the individual does not move outside the region during the next $n-1$ years, while the term following the brackets is the probability that the individual moves in year n. Finally, the 1-year probability, $p_{k,1}$, is computed as the number of individuals age k who move out of the region in a single year divided by the number of individuals age k who lived in the region at the beginning of the year.

Data from the 1990 In-Migration portion of the County-to-County Migration Files published by the U.S. Census Bureau were used to compute the 1-year move probabilities. For each of a series of age groups (ages 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85+), those files quantify the number of current (1990) residents in every U.S. county who have resided in that county during the preceding 5 year period (1985 to 1989), and the number of current residents who moved into the county during the preceding 5-year period. For the latter group, the data quantify how many residents came from each outside county.

In order to estimate the probability of moving into or out of the Upper Hudson counties, the following census information was used:

1. The number of individuals in 1990 who had resided within the five counties since 1985;

2. The number of individuals in 1990 who had moved to their current residence from one of the other four counties within the same Upper Hudson counties; and

3. The number of individuals in 1990 who had moved to their current residence from a county outside the Upper Hudson counties.

The sum of the first and second categories is the number of individuals in 1990 who had been living within that region during the preceding 5 years.

If the age categories divide the population into 5-year increments, then it is by definition true that

$$Start_{1985\text{-}90,k} + In_{1985\text{-}90,k} - Out_{1985\text{-}90,k} = End_{1985\text{-}90,k+1} \quad [3\text{-}5]$$

where

$End_{1985\text{-}90,k+1}$ = Number of individuals in age category $k+1$ at the end of the 1985 to 1990 period.

$Start_{1985\text{-}90,k}$ = Number of individuals in age category k who lived in the region at the beginning of the 1985-1990 period.

$In_{1985\text{-}90,k}$ = Number of individuals in age category k who moved into the region during the past 5 years.

$Out_{1985\text{-}90,k}$ = Number of individuals in age category k who moved out of the region during the past 5 years.

The In-Migration files do not report the value of $End_{1985\text{-}90,k}$. However, under the assumption that the populations in the Upper Hudson counties are in steady state, the number of individuals in age category k at the beginning of the 1985 time period is equal to the number of individuals in the same age category at the end of that time period. Hence, $End_{1985\text{-}90,k+1}$ is assumed to equal $Start_{1985\text{-}90,k+1}$, and Equation [3-5] can be rewritten,

$$Start_{1985\text{-}90,k} + In_{1985\text{-}90,k} - Out_{1985\text{-}90,k} = Start_{1985\text{-}90,k+1} \quad [3\text{-}6]$$

From Equation [3-6], the value of $Out_{1985\text{-}90,k}$ can be calculated as,

$$Out_{1985\text{-}90,k} = (Start_{1985\text{-}90,k} - Start_{1985\text{-}90,k+1}) + In_{1985\text{-}90,k} \quad [3\text{-}7]$$

Finally, the probability that an individual in age category k moves out of the region during a five-year period, denoted $p(k)$, is computed as:

$$p(k) = \frac{Out_{1985-90,k}}{Start_{1985-90,k} + In_{1985-90,k}}$$

Two computational issues must be noted. First, 1-year move probabilities cannot be directly computed using the In-Migration data because the data reflect mobility over a 5-year time period. The number of individuals moving out of an area in a single year were assumed to equal the number who move out over a 5-year time period divided by 5. The 1-year move probabilities were applied to all ages within category k. Second, because the age categories for ages 35 or above are reported in 10-year increments, while those for ages 34 and below are reported in 5-year increments, one-half the value reported for $Start_{1985\text{-}90,35\text{-}44}$ was used in the computation of $Out_{1985\text{-}90,30\text{-}34}$.

Tables 3-8 through 3-12 detail the In-Migration data for each of these five counties separately, and Table 3-13 summarizes the counts summed over these five counties. Table 3-14 lists the values used to compute the 1-year move probabilities, and Table 3-15 provides an overall summary of the move probabilities. Figure 3-5a provides a frequency histogram of the residence duration. The overall frequency distribution for total exposure duration (the combination of fishing duration probability and residence duration probability) is shown in Figure 3-5b.

Assumptions for Residence Duration Estimates

Two basic assumptions were made here in order to estimate the probability distribution of residence duration (and likelihood of moving out of the five counties):

- The population's age distribution was assumed to be at steady-state, and does not change over time.

- The probability that an individual moves was assumed to depend only on his or her current age and not on the length of time he or she has already lived in the area. If the conditional probability of moving out of the area is lower for individuals who have already lived in the area for a long period of time, it is possible that the approach adopted will underestimate the fraction of the population whose residence times are very long.

It is of course likely that the population is not strictly at steady state. However, an adjustment for non-steady state conditions is not apparent, because it would require projecting future trends with historical data. Forecasting future trends was deemed to be a greater source of uncertainty than the necessary assumption of steady state.

The exposure duration distribution ranges from 10 years to 60 years, with a 50^{th} percentile value of 12 years, and a 95^{th} percentile value of 40 years. For comparison, current USEPA recommendations for the exposure duration parameter for Superfund risk assessments are 9 years (median) and 30 years based on population mobility statistics for the general public (USEPA, 1997f). While there are uncertainties inherent in the derivation of the exposure duration for this HHRA, the values are reasonable when compared to national mobility statistics, and also cover the possibility of extended exposure, as long as 60 years, consuming fish from the Upper Hudson River.

3.2.5 Body Weight

The probability distribution of the variation of body weight within the population was drawn from published studies of adult and child/adolescent body weights. Brainard and Burmaster (1992) report that the body weight distributions for males between the ages 18 and 74 years and for females between the ages of 18 and 74 are lognormal. The Brainard and Burmaster (1992) results and the calculated lognormal distribution summary statistics appear in Table 3-16.

Finley *et al.*(1994) report the arithmetic means ($\bar{x}$) and arithmetic standard deviations (s_x) of the body weight distributions for individuals aged 1 to 18 years, and for all individuals greater than 18 years of age. Because the authors do not specify the form of these distributions, they are assumed to be lognormal

based on the lognormality of the adult body weights found by Brainard and Burmaster (1992). Assuming a lognormal distribution of body weight, the geometric mean (GM) and geometric standard deviation (GSD) can be calculated from their arithmetic counterparts by,

$$GM = \exp\left(\ln \bar{x} - GSD^2/2\right)$$

$$GSD = \exp\left(\sqrt{\ln\left(1 + s_x^2/\bar{x}^2\right)}\right)$$

Because body weights can be measured very accurately and the distribution of body weights in the population has been extensively studied and well characterized (*e.g.*, by Finley *et al.* (1994) and Brainard and Burmaster (1992)), the uncertainty associated with this parameter's estimate is likely to be negligible. No sensitivity analysis was deemed necessary for this parameter.

It was assumed that for each individual in the population, body weight is perfectly correlated over time. That is, individuals whose body weight is high at one age will have a high body weight at other ages, while those whose body weight is low at one age will have a low body weight at other ages. To implement this temporal correlation, each simulated individual was assigned a weight distribution percentile, and this body weight percentile was assigned to the simulated individual throughout the exposure duration. For example, the individual who has the median population body weight at age 1 was assigned the median population body weight during the remainder of his or her simulated lifetime, ensuring that individual body weights in the population are correlated over time.

3.3 Summary of Simulation Calculations

The Monte Carlo exposure calculation sequence is shown in Figure 3-1. Each simulation consisted of 10,000 samples, where each sample represents a simulated angler. A summary of the base case and sensitivity analysis distribution inputs is provided in Section 3.3.1. Section 3.3.2 summarizes the numerical stability of the Monte Carlo calculations. The risk estimates that correspond to the Monte Carlo exposure analysis are presented in Chapter 5, following the discussion of PCB toxicity factors in Chapter 4.

3.3.1 Input Distributions Base Case and Sensitivity Analysis

As described above, the Monte Carlo exposure analysis was conducted to examine the RME for the fish ingestion pathway. The probability distributions derived for this analysis are aimed at determining the variability of exposure among the angler population. Throughout the derivation of the input distributions, a recognition of the uncertainty involved in estimating the distributions has been presented. Because insufficient information is available to characterize the uncertainty by means of a fully 2-D Monte Carlo analysis, a sensitivity/uncertainty analysis was performed as an alternative means to address the approximate precision of the analysis.

The sensitivity analysis involved repeating the Monte Carlo analysis for separate input distributions for each of the variable parameters. The 72 combinations evaluated included the following:

*Parameter**	*Base Case*	*Sensitivity Analysis*
Fish Ingestion (4)	1991 New York Angler Survey Empirical Ingestion Distribution	1992 Maine Angler (Ebert *et al.*, 1993) 1989 Michigan (West *et al.*, 1989) 1992 Lake Ontario (Connelly *et al*, 1996)
Exposure Duration (2)	Minimum of Fishing Duration and Residence Duration	Residence Duration only
Fishing Location (3)	Average of 3 Modeled Locations	Thompson Island Pool Waterford/Federal Dam
Cooking Loss (3) (no variability modeled)	20% (midpoint of typical range)	0% (high-end exposure) 40% (low-end exposure)

**Numbers in parentheses indicate number of combinations*

The Monte Carlo exposure analysis examines variability (and sensitivity/uncertainty) only of PCB intake. The intake is translated into health risk by combining the intake results with PCB toxicity factors for both cancer and non-cancer evaluations. Thus, the intake results are scaled linearly by the corresponding toxicity factors. A discussion of the base case Monte Carlo analysis results is presented in Section 5.2 and the sensitivity analysis is discussed in Section 5.3.3.

3.3.2 Numerical Stability Analysis

The Monte Carlo simulations were implemented using SAS version 6.12.[13] A total of 10,000 iterations were performed for each of the 72 scenarios evaluated.

In order to investigate the numerical stability of the Monte Carlo calculations, 100 independent trials, each of 10,000 iterations, were run. As shown below, the small coefficients of variation, which is the standard deviation (s_x) divided by the mean ($\bar{x}$), for various PCB intake percentiles shows that 10,000 samples is sufficient to produce stable numerical results.

Numerical Stability Results (100 Simulations of 10,000 iterations)	
Statistic	*Coefficient of Variation* ($s_x/\bar{x}$)
5th percentile	2.9%
25th percentile	2.3%
50th percentile	1.9%
90th percentile	2.6%
95th percentile	3.8%
99th percentile	6.0%

At the 50th percentile (median) intake, the standard deviation of the 100 simulations (each consisting of 10,000 simulated anglers) was within 1.9% of the mean. For the tails of the intake estimates, the standard

[13] Cohen *et al.* (1996) describe the implementation of a 2-D Monte Carlo simulation using SAS software.

deviation of the 95th percentile intake was within 3.8% of the mean, and for the 99th percentile within 6% of the mean.

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4 Toxicity Assessment

PCBs are a group of synthetic organic chemicals that contain 209 individual chlorinated biphenyl compounds (also known as congeners) with varying harmful effects. There are no known natural sources of PCBs in the environment. PCBs enter the environment as mixtures containing a variety of individual components (congeners) and impurities that vary in toxicity. Commercially available PCB mixtures are known in the U.S. by their industrial trade name, Aroclor. The name, Aroclor 1254, for example, means that the molecule contains 12 carbon atoms (the first 2 digits) and approximately 54% chlorine by weight (second 2 digits). The manufacture processing and distribution in commerce of PCBs in the U.S. was restricted beginning in October 1977 because of evidence that PCBs build up in the environment and cause harmful effects (USEPA, 1978).

At sufficient dose levels, PCBs have been demonstrated to cause a variety of adverse health effects, both carcinogenic and noncarcinogenic. These health effects include cancer, liver toxicity, reproductive toxicity, immunotoxicity, dermal toxicity, and endocrine effects as described in USEPA's IRIS toxicity profiles (USEPA, 1999a-c) and reviewed by Safe (1994) and ATSDR (1997). The toxicity of PCBs for both cancer and non-cancer health effects is summarized in more detail in Appendix C.

USEPA has classified PCBs as "B2" probable human carcinogens based on liver tumors in female rats exposed to Aroclor 1260, 1254, 1242, and 1016, and in male rats exposed to Aroclor 1260 and suggestive evidence from human epidemiological data (USEPA, 1999c). USEPA has also derived reference doses for Aroclors 1016 and 1254 based on non-cancer effects, such as reduced birth weight (Aroclor 1016) and impaired immune function, distorted finger and toe nail beds, and occluded Meibomian glands located in the eyelid (Aroclor 1254).

It is also important to recognize that commercial PCBs tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain (*i.e.*, laboratory test animals were fed Aroclor mixtures, not environmental mixtures that had been bioaccumulated). Bioaccumulated PCBs appear to be more toxic than commercial PCBs and appear to be more persistent in the body (USEPA, 1999c).

Potential non-cancer hazards and cancer risks posed by exposure to PCBs are evaluated using toxicity values, which are determined from systemic toxicity for non-cancer health effects (oral Reference Doses, or RfDs), or chemical dose-response relationships for carcinogenicity (Cancer Slope Factors, or CSFs). Following a rigorous peer review process, the profiles presented in USEPA's Integrated Risk Information System (IRIS) database summarize the toxicity of the individual chemicals.

4.1 Non-cancer Toxicity Values

The chronic RfD represents an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. USEPA derives RfDs by first identifying the highest dose level that does not cause observable adverse effects (the no-observed-adverse-effect-level, or NOAEL). If a NOAEL was not identified, a lowest-observed-adverse-effect-level, or LOAEL, may be used. This dose level is then divided by uncertainty

factors to calculate an RfD. There are four standard uncertainty factors that can be used when calculating an RfD:

- An up-to-10-fold factor to account for the variation in sensitivity among members of the human population.
- An up-to-10-fold factor to account for the uncertainty involved in extrapolating from animal data to humans.
- An up-to-10-fold factor to account for the uncertainty involved in extrapolating from less than chronic NOAELs to chronic NOAELs.
- An up-to-10-fold factor to account for the uncertainty involved in extrapolating from LOAELs to NOAELs.

An additional modifying factor can also be applied to the calculation of the RfD. The modifying factor is an additional uncertainty factor that is greater than zero and less than or equal to 10. The magnitude of the modifying factor depends upon an assessment of the scientific uncertainties of the study and the database used in deriving the RfD that are not explicitly treated above; *e.g.*, completeness of the overall data base and number of species tested.

The IRIS database provides oral RfDs for two Aroclor mixtures, Aroclor 1016 and Aroclor 1254. There is no RfD available for Total PCBs (Table 4-1) and Aroclor 1248. The RfD for Aroclor 1016 is 0.00007 (7×10^{-5}) mg/kg-day, based on the NOAEL for reduced birth weight in a monkey reproductive bioassay, and an uncertainty factor of 100. This RfD is more stringent than the former RfD of 0.0004 used in the Phase 1 risk assessment.

The RfD for Aroclor 1254 is 0.00002 (2×10^{-5}) mg/kg-day, based on the LOAEL for impaired immune function, distorted finger and toe nail beds, and occluded Meibomian glands in the rhesus monkey, and an uncertainty factor of 300.

For both Aroclor 1016 and Aroclor 1254, the USEPA reports "medium" confidence in the toxicity studies on which the RfDs are based, the overall toxicity database, and the RfDs themselves.

Although there is an IRIS file for Aroclor 1248, the USEPA determined the available health effects data to be inadequate for derivation of an oral RfD (USEPA, 1999e). However, a brief summary of the principal findings of animal studies is included in the IRIS file (USEPA, 1999d). Results of the studies showed impairment of reproduction in female rhesus monkeys lasting more than 4 years after dosing, reduced birth weight for infants, facial acne and edema, swollen eyelids, and hair loss.

Due to various environmental processes, PCB mixtures present in the environment no longer resemble the Aroclor mixture originally released into the environment. Therefore, although the General Electric Company facilities historically used primarily Aroclor 1242 in their operations, the PCBs present in Upper Hudson River fish, sediment, and river water do not have the same distribution of PCB congeners

as any of the commercial Aroclor mixtures. However, since RfD values are only available for Aroclor mixtures and not Total PCBs, it was necessary to choose the Aroclor mixture most similar to the PCBs present in Upper Hudson River fish, sediment, and river water.

The PCB homologue distribution of sediment and water samples is predominately dichloro- through pentachlorobiphenyls, as reported in the Hudson River Data Evaluation and Interpretation Report (USEPA, 1997d). This distribution is more similar to Aroclor 1016 than to Aroclor 1254. Therefore, for the purposes of this HHRA, PCBs in sediment and water samples were considered to be most like Aroclor 1016. The Aroclor 1016 RfD (7×10^{-5} mg/kg-day) was used to evaluate non-cancer toxicity for ingestion of Upper Hudson River sediment, dermal contact with Upper Hudson River sediment, and dermal contact with Upper Hudson River water.

The PCB homologue distribution in fish differs from the sediment and water samples due to differential bioaccumulation of PCB congeners with higher chlorination levels. Trichloro- through hexachlorobiphenyls contribute to the majority of fish tissue PCB mass as reported in the Baseline Modeling Report (USEPA, 1999d). This distribution is more similar to Aroclor 1254 than to Aroclor 1016. Therefore, for the purposes of this HHRA, PCBs in fish were considered to be most like Aroclor 1254. The Aroclor 1254 RfD (2×10^{-5} mg/kg-day) was used to evaluate non-cancer toxicity for ingestion of Upper Hudson River fish for both the point estimate and probabilistic assessments. Consistent with USEPA policy (USEPA, 1997a), uncertainty and variability in the toxicity values are not quantitatively evaluated in the Monte Carlo analysis.

The Aroclors tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain. For exposure through the food chain, therefore, health hazards can be higher than those estimated in this assessment.

As indicated in Table 4-2, there are no Reference Concentrations (RfCs) currently available for either Total PCBs or any of the Aroclor mixtures (USEPA, 1999a-c). Therefore, inhalation exposures to PCBs are evaluated only for cancer (using the CSF), and not for non-cancer effects.

4.2 PCB Cancer Toxicity

The Cancer Slope Factor, or CSF, is a plausible upper bound estimate of carcinogenic potency used to calculate risk from exposure to carcinogens, by relating estimates of lifetime average chemical intake to the incremental risk of an individual developing cancer over a lifetime. The CSFs developed by the USEPA are plausible upper bound estimates, which means that the USEPA is reasonably confident that the actual cancer risk will not exceed the estimated risk calculated from the CSF.

USEPA has classified PCBs as "B2" probable human carcinogens based on liver tumors in female rats exposed to Aroclor 1260, 1254, 1242, and 1016, and in male rats exposed to Aroclor 1260 and suggestive evidence from human epidemiological data (USEPA, 1999c). In IRIS, which summarizes the Agency's review of toxicity data (USEPA, 1999a-c), both upper-bound and central-estimate CSFs are listed for three different tiers of PCB mixtures (Aroclor 1260, 1254, 1242, and 1016). These PCB mixtures contain overlapping groups of congeners that span the range of congeners most often found in environmental mixtures. The CSFs are based on the USEPA's reassessment of the toxicity data on the potential carcinogenic potency of PCBs in 1996 (USEPA, 1996b; Cogliano, 1998) and were derived

following the proposed revisions to the USEPA Carcinogen Risk Assessment Guidelines (USEPA, 1996b), including changes in the method of extrapolating from animals to humans and changes in the categories for classifying the carcinogenic potential of chemicals. The CSF reassessment was also externally peer-reviewed. The first tier, "High Risk and Persistence," applicable to food chain exposures, sediment or soil ingestion, dust or aerosol inhalation, dermal exposure, early-life exposure, and mixtures with dioxin-like, tumor promoting, or persistent congeners, has upper-bound and central-estimate CSFs of 2.0 and 1.0 $(mg/kg\text{-}day)^{-1}$, respectively. The second tier, "Low Risk and Persistence," applicable to ingestion of water-soluble congeners, inhalation of evaporated congeners, and dermal exposure (if no absorption factor has been applied), has upper-bound and central-estimate CSFs of 0.4 and 0.3 $(mg/kg\text{-}day)^{-1}$, respectively. The third tier, "Lowest Risk and Persistence," applicable only to mixtures where congeners with more than four chlorines comprise less than one-half percent of the Total PCBs, has upper-bound and central-estimate CSFs of 0.07 and 0.04 $(mg/kg\text{-}day)^{-1}$, respectively.

The Aroclors tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain. For exposure through the food chain, therefore, risks can be higher than those estimated in this assessment.

Consistent with the recommended values in IRIS, the first tier upper-bound and central-estimate CSFs of 2.0 and 1.0 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central-estimate exposures to PCBs via ingestion of Upper Hudson River fish, ingestion of Upper Hudson River sediments, and dermal contact with Upper Hudson River sediments (Table 4-3). These CSFs are lower than the former value of 7.7 $(mg/kg\text{-}day)^{-1}$ used in the Phase 1 risk assessment as a result of new scientific data and changes in the methods for calculating the CSF as indicated in the proposed Carcinogen Guidelines (USEPA, 1996b). The second tier upper-bound and central-estimate CSFs of 0.4 and 0.3 $(mg/kg\text{-}day)^{-1}$ are used to evaluate cancer risks for the upper-bound and central-estimate exposures to PCBs *via* dermal contact with Upper Hudson River water and potential inhalation of PCBs volatilized from the Upper Hudson River (Tables 4-3 and 4-4). In the Phase 1 risk assessment, the former CSF value of 7.7 $(mg/kg\text{-}day)^{-1}$ was used.

For the Monte Carlo analysis of cancer risks *via* fish ingestion, only the upper bound CSF of 2.0 $(mg/kg\text{-}day)^{-1}$ is used. Consistent with USEPA policy (USEPA, 1997a), variability and uncertainty in chemical toxicity is not quantitatively evaluated in the Monte Carlo analysis.

4.3 Toxic Equivalency Factors (TEFs) for Dioxin-Like PCBs

A subset of PCB congeners are considered to be dioxin-like, that is, they are structurally similar to dibenzo-p-dioxins, bind to the aryl hydrocarbon receptor, and cause dioxin-specific biochemical and toxic responses (reviewed in USEPA, 1996b). Several investigators have estimated the carcinogenic potency of these dioxin-like PCB congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

Dr. Safe proposed TEFs for a number of dioxin-like PCBs based on a review of the available scientific data on the toxicity and mechanisms of action of dibenzo-p-dioxin, dibenzofuran, and PCB congeners (Safe, 1990; Safe, 1994). In 1994, the World Health Organization (WHO) European Center for Environment and Health and the International Program on Chemical Safety (IPCS) published recommended interim TEFs for thirteen dioxin-like PCB congeners based on a comprehensive review of the available scientific literature and consultation with twelve international PCB experts (Ahlborg *et al.*,

1994). The 1994 WHO/IPCS TEFs are summarized in Table 4-5. In 1996, USEPA recommended that the 1994 WHO/IPCS TEFs could be used to supplement analyses of PCB carcinogenicity (USEPA, 1996c). Subsequently, WHO/IPCS held a meeting in 1997 to reevaluate and update TEFs for dioxin-like PCBs (Van den Berg *et al.*, 1998) based on a review of both previously reviewed and new data. Their revised TEFs for human health risk assessment were published in 1998 and are also summarized in Table 4-5. Only four TEFs were changed: the TEF for PCB congener 77 was reduced from 0.0005 to 0.0001, a TEF for congener 81 was added, and the TEFs for congeners 170 and 180 were withdrawn.

Dioxin-like PCB congeners are responsible for only part of the carcinogenicity of a Total PCB mixture. To account for the fact that relative concentrations of dioxin-like congeners may be enhanced in environmental mixtures, particularly in fish due to bioaccumulation of more persistent congeners, the 1998 WHO/IPCS TEFs are used in the risk characterization, along with the CSF of 150,000 $(mg/kg\text{-}day)^{-1}$ for dioxin, to supplement the evaluation of PCB cancer risks due to consumption of fish (HEAST, 1997). (Note that use of the 1994 WHO/IPCS TEFs would result in similar risk estimates.)

4.4 Endocrine Disruption

In response to growing concerns about the potential effects of environmental endocrine disruptors on human health, the USEPA's Risk Assessment Forum held several workshops to discuss the current status of knowledge on endocrine disruption at the request of the USEPA Science Policy Council in 1997. As a result of these workshops, USEPA prepared the "Special Report on Environmental Endocrine Disruption: An Effects Assessment and Analysis" (USEPA, 1997b) which is intended to inform Agency risk assessors of the major findings and uncertainties and to serve as a basis for a Science Policy Council position statement.

An environmental endocrine disruptor is defined as "an exogenous agent that interferes with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, development, and/or behavior" (USEPA, 1997b, pg. 1).

PCBs have been investigated as potential endocrine disruptors. For example, some studies have suggested that PCBs increase the risk of breast cancer, while other studies have failed to show an association between PCB exposure and breast cancer (reviewed in USEPA, 1997b). Overall, the USEPA Risk Assessment Forum concluded that it is not possible to attribute a cause and effect association between PCB exposure and breast cancer given the sparse data currently available. Similarly, an association between endometriosis and high levels of PCBs in blood has been reported, but the evidence for a causal relationship is considered weak (reviewed in USEPA, 1997b). Due to the similar structural properties of PCBs and normal thyroid hormones (T_4 and T_3), PCBs may also cause thyroid effects such as hypothyroidism (reduction of thyroid hormones in circulation) *via* competition for receptor binding (reviewed in USEPA, 1997b). The mechanisms of thyrotoxicity associated with PCB exposure may vary and include specific damage to the endocrine gland, interference with hormone transport, and receptor interactions (USEPA, 1997b). For example, in rats, prenatal exposure to some PCBs (specific congeners or mixtures such as Aroclor 1254) have been shown to lower serum T_4 which reduces choline acetyl transferase (ChAT) activity in the hippocampus and basal forebrain. ChAT is involved in the synthesis of acetylcholine, a neurotransmitter considered important to learning and memory (USEPA, 1997b). PCB exposures may also be associated with an increase in thyroid follicular cell adenomas or

carcinomas in male rats with a statistically significant trend for Aroclor 1242 and 1254 (Mayes *et al.*, 1998).

There is currently considerable scientific debate about whether environmental chemicals acting *via* endocrine disruptor mechanisms are responsible for adverse health effects in humans (reviewed in USEPA, 1997b). Because the human body has negative feedback mechanisms to control the fluctuations of hormone levels, exposures to chemicals at the levels found in the environment may be insufficient to disrupt endocrine homeostasis. Current screening assays that measure hormone receptor binding thus may or may not be associated with a corresponding adverse health effect. Furthermore, exposures to potential environmental endocrine disruptors are minimal compared to exposures to potential endocrine disruptors that occur naturally in food. However, it is also possible that infants and children are more sensitive to potential endocrine disruptor effects during sensitive windows of development.

The USEPA is aware and concerned about the potential effects of environmental endocrine disruptors on human health, and is currently supporting significant research in this area along with other federal agencies. However, "there is little knowledge of or agreement on the extent of the problem," and "further research and testing are needed" (USEPA, 1997b, pg. vii). The USEPA Science Policy Council's Interim Position is that "based on the current state of the science, the Agency does not consider endocrine disruption to be an adverse endpoint per se, but rather to be a mode or mechanism of action potentially leading to other outcomes, for example, carcinogenic, reproductive, or developmental effects, routinely considered in reaching regulatory decisions" (USEPA, 1997b, pg. viii).

Therefore, consistent with current USEPA policy, although PCBs may act as an environmental endocrine disruptor, the available data are insufficient to support a quantitative assessment of endocrine effects in this risk assessment. Potential adverse health effects resulting from PCBs operating through a potential endocrine disruption mechanism of action is an area of uncertainty.

5 Risk Characterization

Risk characterization is the final step of the risk assessment process, which combines the information from the Exposure Assessment and Toxicity Assessment steps to yield estimated non-cancer hazards and cancer risks from exposure to PCBs. In addition, risk characterization involves an evaluation of the uncertainties underlying the risk assessment process, and this evaluation is included in this section. The risk characterization was prepared in accordance with USEPA guidance on risk characterization (USEPA, 1995b; USEPA, 1992b).

In Section 5.1, the point estimate calculations of non-cancer hazard indices and cancer risks are presented. The Monte Carlo risk estimates for the base case analysis are summarized in Section 5.2. A discussion of uncertainties inherent to the exposure and toxicity assessments is presented in Section 5.3, along with a quantitative evaluation of the uncertainty in risk characterization for the fish ingestion pathway.

5.1 Point Estimate Risk Characterization

5.1.1 Non-cancer Hazard Indices

The evaluation of non-cancer health effects involves a comparison of average daily exposure levels with established Reference Doses (RfDs) to determine whether estimated exposures exceed recommended limits to protect against chronic adverse health hazards. A Reference Dose is defined as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound, with chronic duration ranging from seven years to a lifetime as a Superfund guideline (USEPA, 1989b).

Potential health hazards from noncarcinogenic effects are expressed as a Hazard Quotient (HQ), which compares the calculated exposure (average daily doses, calculated as part of the exposure assessment in Chapter 2) to the RfD (summarized as part of the toxicity assessment in Chapter 4). Both exposure levels and RfDs are typically expressed in units of mass of PCB intake per kilogram of body weight per day (mg/kg-day). Unlike the evaluation of carcinogenic effects, exposures of less than lifetime duration are not averaged over an entire lifetime but rather the duration of exposure (USEPA, 1989b).

The hazard quotient is calculated by dividing the estimated average daily oral dose estimates by the oral RfD as follows (USEPA, 1989b):

$$Hazard\ Quotient\ (HQ) = \frac{Average\ Daily\ Dose\ (mg/kg-day)}{RfD\ (mg/kg-day)} \qquad [5\text{-}1]$$

High-end and central tendency hazard quotients calculated for each exposure pathway (fish ingestion, sediment, and water exposure pathways) are summarized in Tables 5-1 through 5-13. Hazard Quotients are summed over all COPCs (chemicals of potential concern) and all applicable exposure routes

to determine the total Hazard Index (HI). In this HHRA, PCBs are the COPCs and the HQ for PCBs is equivalent to the HI. The total high-end and central tendency Hazard Indices for each pathway and receptor are summarized in Tables 5-27 through 5-33.

If a Hazard Index is greater than one (*i.e.*, HI>1), unacceptable exposures may be occurring, and there may be concern for potential non-cancer effects, although the relative value of an HI above one (1) cannot be translated into an estimate of the severity of the hazard. Ingestion of fish results in the highest Hazard Index, with an HI of 10 for the central tendency estimate, and an HI of 116 for the high-end estimate, both representing exposures above the reference level (HI>1). Note that as discussed earlier, the average daily dose decreases as the exposure duration increases, so the average concentration over a 7-year exposure period (used as the high-end estimate in this HHRA) is greater than the average concentration over the RME duration of 40 years. Even if the average concentration over a 40-year exposure period is used (*i.e.*, 2.2 ppm instead of 5.1 ppm), a hazard index of 50 results, which is still above the reference level of 1. Total Hazard Indices for the recreational and residential exposure pathways are all below one. In all cases, the Hazard Indices are based on uniform exposure throughout the Upper Hudson River. Uncertainties inherent in these risk estimates are discussed later in this report.

5.1.2 Cancer Risks

Cancer risks are characterized as the incremental increase in the probability that an individual will develop cancer during his or her lifetime due to site-specific exposure. The term "incremental" implies the risk due to environmental chemical exposure above the background cancer risk experienced by all individuals in the course of daily life. Cancer risks are expressed as a probability (*e.g.*, one in a million, or 10^{-6}) of an individual developing cancer over a lifetime, above background risk, as a result of exposure.

The quantitative assessment of carcinogenic risks involves the evaluation of lifetime average daily dose and application of toxicity factors reflecting the carcinogenic potency of the chemical. Specifically, excess (incremental) cancer risks are calculated by multiplying intake estimates (lifetime average daily doses, calculated in Chapter 2 as part of the exposure assessment) and CSFs (summarized as part of the toxicity assessment in Chapter 4) as follows (USEPA, 1989b):

$$Cancer\ Risk = Intake\left(\frac{mg}{kg-day}\right) \times CSF\left(\frac{mg}{kg-day}\right)^{-1} \qquad [5\text{-}2]$$

As discussed in Chapter 2, exposure levels are expressed as the chronic daily intake averaged over a lifetime of exposure, in units of mg/kg-day (mg of PCB intake per kilogram of human body weight per day). A cancer slope factor is an estimate of the upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level or dose of a potential carcinogen. Cancer slope factors are expressed in units that are the reciprocal of those for exposure (*i.e.*, $(mg/kg\text{-}day)^{-1}$). Multiplication of the exposure level by the CSF yields a unitless estimate of cancer risk. The acceptable risk range identified in the NCP (USEPA, 1990) is 10^{-4} to 10^{-6} (or an increased probability of developing cancer of 1 in 10,000 to 1 in 1,000,000) refers to plausible upper bound risks.

High-end and central tendency cancer risk estimates calculated for each exposure pathway (fish ingestion, recreational exposure pathways, and residential inhalation) are summarized in Tables 5-14

through 5-26. Total cancer risks are summed over all applicable exposure routes and exposure periods (child through adult). The total RME and central tendency cancer risks for each pathway are summarized in Tables 5-27 through 5-33.

Ingestion of fish results in the highest cancer risks, 3.2×10^{-5} (3.2 additional cases of cancer in a population of one hundred-thousand) for the central tendency estimate, and 1.1×10^{-3} (1.1 additional cancers in a population of a thousand) for the high-end estimate. Risks for children consuming fish were included in the Monte Carlo exposure calculations, however they cannot be specifically identified in the Monte Carlo results because those results are for the entire population of anglers. If it is assumed that a child meal portion is approximately of an adult portion, then the RME child risk for ingestion of fish is approximately 3×10^{-4}. As a further note on the fish ingestion risks, had the 95th percentile fish ingestion rate (63.4 g/day, or 102 meals per year) been used in the analysis, the RME risks for fish ingestion would approximately double (*i.e.*, 2×10^{-3} for adults).

As indicated earlier, the acceptable cancer risk range established in the NCP is 10^{-4} to 10^{-6}. Thus, the RME fish ingestion results fall outside the NCP acceptable cancer risk range. Estimated cancer risks relating to PCB exposure in either sediment, water, or air are much lower than those for fish ingestion, falling generally at the low end, or below, the range of 10^{-4} to 10^{-6}.

5.1.3 Dioxin-Like Risks of PCBs

To account for the fact that relative concentrations of dioxin-like congeners may be enhanced in environmental mixtures, particularly in fish due to bioaccumulation of more persistent congeners, the 1998 WHO/IPCS TEFs are used in the risk characterization, along with the CSF of 150,000 for dioxin (USEPA, 1997), to supplement the evaluation of PCB cancer risks due to consumption of fish.

This analysis was performed using the Phase 2 fish data from the Upper Hudson River (River Miles 159-196.9) contained in the Hudson River database. For each Phase 2 fish sample in the Upper Hudson River, the concentrations total (tri+) PCBs, were summarized (Tables 5-34).[14,15] In order to determine the fraction that each dioxin-like congener represented of the Total PCB concentration, the concentration of each dioxin-like PCB congener was divided by the Total PCB concentration for each fish sample, (Table 5-35). These fractions were averaged over all the fish samples to determine an average fraction for each dioxin-like congener (Table 5-35, last two rows). These fractions were then multiplied by the high-end Total PCB exposure point concentration used in the risk assessment, to determine the high-end exposure point concentration for each dioxin-like congener (Table 5-36). These exposure point concentrations were then multiplied by the corresponding 1998 WHO/IPCS toxicity equivalency factors TEF to generate a dioxin equivalent (TEQ) for each dioxin-like congener (Table 5-36 last column). The TEQs for each congener were summed, yielding a high-end total dioxin TEQ of 5.3×10^{-5} mg/kg (Table 5-36, second to last row). The total concentration of the non-dioxin-like PCB congeners was calculated

[14] Note that although PCB congener 81 is considered a dioxin-like PCB congener, it was not analyzed for as part of the analytical program. At the time the analytical sampling methods were determined for the Phase 2 program, a standard for congener 81 was unavailable. The risks for this congener are not included in this risk analysis.

[15] Non-detect values were set to ½ the detection limit if the total detection frequency was greater than 15% (based on professional judgment) for that congener. If the total detection frequency was less than 15%, the value was set to zero.

by subtracting the sum of the concentrations of the dioxin-like congeners from the high-end Total PCB exposure point concentration (Table 5-36, last row).

Cancer risks for ingestion of dioxin-like PCBs in fish were calculated similarly to those for PCBs, substituting the dioxin TEQ for the exposure point concentration and the dioxin CSF of 150,000 (USEPA, 1997) for the cancer slope factor. The resulting intake and cancer risk estimates are shown in Table 5-38. The RME dioxin-like cancer risk of 1.5×10^{-3} is approximately equivalent to the RME risk calculated without consideration of the dioxin-like congeners, and, similarly, is outside of the acceptable range for cancer risk established in the NCP.

5.2 Monte Carlo Risk Estimates for Fish Ingestion

As described in Section 3.5.1, a total of 72 scenarios were evaluated for the Monte Carlo exposure analysis. The non-cancer hazards and cancer risk estimates for each scenario were calculated using the same equations outlined in Sections 5.1.1 and 5.1.2, respectively, using Equation [3-1] to calculate PCB intake. The combination of scenarios discussed in Section 3.5.1 is reproduced here for convenience:

Parameter*	***Base Case***	***Sensitivity Analysis***
Fish Ingestion (4)	1991 New York Angler Survey Empirical Ingestion Distribution	1992 Maine Angler (Ebert *et al.*, 1993) 1989 Michigan (West *et al.*, 1989) 1992 Lake Ontario (Connelly *et al*, 1996)
Exposure Duration (2)	Minimum of Fishing Duration and Residence Duration	Residence Duration only
Fishing Location (3)	Average of 3 Modeled Locations	Thompson Island Pool Waterford/Federal Dam
Cooking Loss (3) (no variability modeled)	20% (midpoint of typical range)	0% (high-end exposure) 40% (low-end exposure)

**Numbers in parentheses indicate number of combinations*

5.2.1 Non-Cancer Hazards

For the non-cancer hazard calculations, *Average Daily Dose* in Equation [5-1] was calculated using Equation [3-1], with a maximum exposure duration (ED in Equation [3-1]) of 7 years. This exposure duration limit was selected as the minimum time-period for chronic exposure. Because the Average Daily Dose declines as the exposure duration increases, allowing the intake to be averaged over a longer time-period would underestimate non-cancer hazards and potentially underestimate the hazard for an RME individual.[16]

Each of the 72 scenarios examined consisted of 10,000 simulations of PCB intake (average daily dose), each yielding a distribution of 10,000 intake estimates. From these distributions of intake, low-end, mid-point, and high-end non-cancer hazard index percentiles (5^{th},..., 50^{th}, 90^{th}, 95^{th}, 99^{th}) are summarized in Appendix B.

[16] The dependency of the intake on ED is due to the time-dependency of PCB concentration in fish.

A relative frequency and cumulative distribution plot for the "base case" analysis is shown in Figure 5-1a. The median HI for the base case Monte Carlo analysis is 11.4, compared with the HI of 10 for the central point estimate. The 95th percentile HI from the base case Monte Carlo analysis is 137, compared with 116 for the RME point estimate. At the high-end of the base case hazard distribution, the 99th percentile HI is 639; at the low end, the 5th percentile HI is 1.2, and the 10th percentile HI is 1.9.

The Monte Carlo analysis of non-cancer hazards is discussed further in the discussion of uncertainties later in Section 5.3.3.

5.2.2 Cancer Risks

For the cancer risk calculations, *Intake* in Equation [5-2] was calculated using Equation [3-1]. In the case of cancer risks, intake is averaged over a lifetime such that ED in Equation [3-1] was not limited to 7 years, but rather equaled the particular ED value that was sampled from the input probability distribution for this variable on each of the 10,000 iterations.

As was the case for non-cancer hazards, each of the 72 scenarios examined consisted of 10,000 simulations of PCB intake, resulting in a distribution of 10,000 intake estimates. From these distributions of intake, low-end, mid-point, and high-end cancer risk percentiles (5th,..., 50th, 90th, 95th, 99th) are summarized in Appendix B.

A relative frequency and cumulative distribution plot for the "base case" analysis is shown in Figure 5-2a. The median cancer risk for the base case Monte Carlo analysis is 6.4×10^{-5}, which is 2-fold higher than the central point estimate value of 3.2×10^{-5}. The 2-fold difference of these two estimates is directly tied to the fact that the PCB cancer slope factor used for the Monte Carlo estimate (2.0 mg/kg-day^{-1}) is 2-fold greater than the CSF used for the central point estimate (1.0 mg/kg-day^{-1}). The 95th percentile cancer risk estimate for the base case Monte Carlo analysis is 8.7×10^{-4} , compared with 1.1×10^{-3} for the RME point estimate. At the high-end of the base case cancer risk distribution, the 99th percentile is 3.7×10^{-3}; at the low end, the 5th percentile is 5.5×10^{-6}, and the 10th percentile 9.6×10^{-6}.

The Monte Carlo analysis of cancer risk is discussed further in the discussion of uncertainties later in Section 5.3.3.

5.3 Discussion of Uncertainties

The process of evaluating human health risks involves multiple steps. Inherent in each step of the process are uncertainties that ultimately affect the final risk estimates. Uncertainties may exist in numerous areas, including environmental PCB concentration data, derivation of toxicity values, and estimation of potential site exposures. In this section, the significant sources of uncertainty in three of the four risk assessment steps (Exposure Assessment, Toxicity Assessment, and Risk Characterization) are qualitatively discussed, including the strengths, limitations, and uncertainties inherent in key scientific issues and science policy choices. This HHRA accounts for sources of uncertainty in the various components of the risk assessment analysis in order to provide a full understanding of the accuracy and reliability of calculated risks and hazards. An understanding of the strengths and potential uncertainties of the risk

assessment provides the risk manager with additional information for consideration in the risk management decision.

5.3.1 Exposure Assessment

Selection of Exposure Pathways. There are some uncertainties inherent in the selection of exposure pathways quantitatively evaluated in the risk assessment. Fish consumption is the most significant source of risk due to exposure to PCBs in the Upper Hudson River. Anglers also may be exposed to PCBs in sediments and surface water while fishing. However, even if the angler experienced incidental ingestion of sediment, dermal contact with sediment and river water, and inhalation comparable to the adult recreator, such exposure would not measurably increase the cancer risk or non-cancer hazard indices because the fish ingestion pathway risks outweigh all others by several orders of magnitude.

As discussed in Section 2.1.3, there were insufficient data to evaluate intake of PCBs *via* ingestion of home-grown crops, beef, dairy products, eggs, *etc*. and these potential exposure pathways were not quantitatively evaluated in the risk assessment. Although the magnitude of the potential risks from these pathways cannot be reliably quantified with available information, the risks are likely to be minimal when compared to those evaluated quantitatively. In addition, evaluation of the inhalation pathway was limited based on the lack of an RfC.

Defining the Angler Population. For the purposes of this risk assessment, the angler population is defined as those individuals who consume self-caught fish from the Hudson at least once per year, in the absence of a fishing ban or health advisories. The start date for the assessment is 1999, the year in which the risk assessment is released. Thus, the risk assessment considers all anglers fishing in the Upper Hudson River from 1999 into the future. Although this population includes anglers who have been fishing for a long period of time, as well as anglers who may have just started fishing, only exposures occurring in 1999 and later were quantified in the risk assessment. The angler population could have alternatively been defined as the subset of anglers who *began* fishing in 1999 (or recently). During the development of the Monte Carlo analysis, intake was modeled both ways. The results were comparable for both the angler population fishing in the Upper Hudson River in 1999, as well as the subset of anglers who were assumed to *begin* fishing in 1999. Based on the similarity of the two analyses, only a single angler population, based on the full set of data from Connelly *et al.* (1992), was used for the exposure duration analysis.

Risks to individuals who move into, or are born into the area after 1999 were not quantitatively evaluated in the risk assessment. Similarly, those individuals consuming Upper Hudson River fish caught by a friend or family member or received as a gift were also not quantitatively evaluated. There is little quantitative information available on such exposures. Nonetheless, the risks for these individuals are expected to be less than the risks for the angler population, because friends and family members of anglers would be expected to have lower fish consumption rates than the angler population evaluated in this risk assessment.

PCB Exposure Concentration in Fish. During Phase 2 of the Reassessment RI/FS, USEPA has expended considerable effort to characterize current and future PCB concentrations in fish. Despite the extensive amount of information developed, there is still some uncertainty in the exposure point PCB concentrations in fish used in the risk assessment. The primary source of PCB concentrations in fish was the 1999 Baseline Modeling Report (USEPA, 1999d). This report provided information about the

variability of predicted PCB concentrations in future years within each modeled fish species. Although there are uncertainties inherent in the modeling approaches (see USEPA, 1999d), there is insufficient quantitative information available about the precise magnitude of the uncertainties to give a quantitative range of risks attributable to model uncertainty. Based on the ability of the fish bioaccumulation models to capture the historical observed PCB measurements in fish, the model uncertainty in PCB projections in fish is not expected to be sufficient to alter the overall conclusions in this risk assessment Furthermore, the sensitivity/uncertainty analysis conducted for the Monte Carlo analysis provides a measure of the range of exposure and risks as a function of two important factors influencing the exposure point concentration: variations in the fish species caught (different species tend to have different characteristic PCB uptake), and variations in fishing location (the concentration trends decline substantially between the upper and lower reaches of the Upper Hudson River).

Because PCB bioaccumulation in fish was only modeled in the Baseline Modeling Report through the year 2018, it was necessary to extrapolate the modeled results to the year 2069 in order to yield a 70-year potential exposure duration for the Monte Carlo analysis. An exponential trend/regression line provided a reasonably good fit for the regressions. It is unlikely that this approach would contribute to significant underestimates of future exposures had the bioaccumulation model been extended further into the future.

Other sources of uncertainty in the PCB concentrations in fish used in the assessment include the fact that concentrations were averaged over location, and weighted by species. While it is likely that different anglers fish in different locations of the Upper Hudson River there is little information available to quantify these differences, and the presence of current fishing restrictions preclude gathering such information. Instead, a sensitivity analysis of the risks associated with a possible population of anglers who fish only in the upstream areas of the Upper Hudson River study area, where PCB concentrations in fish are the highest, is presented in Section 5.3.3, below. Fish species-specific consumption frequencies were estimated based on the 1991 New York Angler survey (Connelly *et al.*, 1992) from which 226 angler responses report consuming self-caught fish. The variability of fish consumption preference was modeled in the Monte Carlo analysis based on the range of species consumption patterns reflected in that survey.

Fish Ingestion Rate. The primary source used to derive the distribution of fish ingestion for the risk assessment was the 1991 New York Angler survey (Connelly *et al.*, 1992). There are numerous uncertainties inherent in the fish ingestion rate assumptions used in the risk assessment, the most significant of which are discussed below. Despite these uncertainties, the assumptions regarding fish consumption are believed to be reasonable and health protective. The sensitivity analysis conducted for this parameter provides a measure of the range of risks using several alternative sources of information regarding sportfish ingestion.

As stated at the outset, the intent of the HHRA was to evaluate risks for Upper Hudson River anglers in the absence of a fishing ban or Hudson-specific health advisories. Because there *are* current advisories to eat no fish from the Upper Hudson River, it is not possible to collect site-specific information about angler activities in the Upper Hudson River in the absence of health advisories. Therefore, it was necessary to select a distribution of fish ingestion rates from survey information other than surveys only of the Hudson. There is some uncertainty as to whether data from flowing waterbodies from the 1991 New York Angler survey (Connelly *et al.*, 1992) accurately represents Upper Hudson River anglers. Although the fish ingestion rates reported in the New York Angler survey are presumably influenced by general,

non-specific NYSDEC fishing regulations (that would be in effect regardless of PCB contamination levels in the Hudson), because the survey was state-wide, it is not likely to be unduly affected by the Hudson-specific health advisories, and thus considered to be a reasonable surrogate for the Upper Hudson.

Of the available studies of sportfish ingestion, the 1991 New York Angler survey (Connelly *et al.*, 1992) is considered the preferred study to represent Upper Hudson River anglers because, among other reasons outlined in this report, it was conducted in New York and included a large sample size. Other New York waterbodies are likely to be more similar to the Hudson than waterbodies in other states. The fact that the fish ingestion rates from the 1991 New York Angler survey are reasonably consistent with the results of published studies investigating freshwater fish ingestion rates from other locations in the U.S. lends an additional degree of confidence in the use of the 1991 New York Angler survey data.

Risks were not specifically quantified for subsistence anglers, unlicensed anglers, or other subpopulations of anglers who may be highly exposed. Although there are no known, distinct subpopulations that may be highly exposed, there is some degree of uncertainty as to whether these subpopulations have been adequately addressed in this risk assessment. However, as discussed in Section 3.2.1.4, based on consideration of fish ingestion rates among low income families (Wendt, 1986), fish ingestion rates reported for licensed and non-licensed anglers from the Hudson angler surveys (Barclay, 1993; NYSDOH, 1999), and fish ingestion rates for angler populations in other areas of the country (see Table 3-2), it seems likely that any highly exposed subpopulations are represented in the upper percentiles of the fish ingestion rate distribution used in the Monte Carlo analysis.

The consumption rate chosen for each angler modeled is assumed to remain the same from year to year; this approach assumes that fish ingestion rates are perfectly correlated each year. Actual year to year ingestion rates are probably correlated to a high degree, but not perfectly (100%). This assumption is supported by the finding that when classified as either low or high avidity (in relation to the median fishing effort), two-thirds of Lake Ontario anglers were classified the same in 1991 and 1992 (Connelly and Brown, 1995). Assuming there is no correlation between yearly ingestion rates would effectively average high-end consumers out of the analysis, and would clearly be inappropriate. Thus, although there are no data available to quantify the correlation between yearly ingestion rates, the approach taken in the risk assessment is reasonable and protective of human health.

While some anglers may consume fish at frequencies less than once per year and some friends or family members of anglers may consume "gift fish" at infrequent intervals, there are no data to quantify the fish ingestion rates for these individuals. Nonetheless, consideration of only those anglers who consume self-caught fish from the Hudson at least once per year is protective of human health, because exposure to less frequent anglers, family members, or friends would be lower than the exposure calculated for the angler population.

Angler Exposure Duration. The distribution of angler exposure durations developed for use in the Monte Carlo assessment represents variability among anglers. The uncertainties inherent in developing the exposure duration of anglers were described in Section 3.2.4. For example, it was assumed that the age profile of the angler population remains unchanged over time, and that 1991 angler data is representative of 1999 anglers. Insufficient information is available to evaluate these sources of uncertainty quantitatively. Nonetheless, the resulting point estimates (*e.g.*, a central tendency estimate of

12 years, and an RME estimate of 40 years) are unlikely to underestimate actual exposure durations significantly.

PCB Cooking Losses. *A*s described in Section 3.2.3, reported cooking losses vary considerably among the numerous studies reviewed. In addition, there is little information available to quantify personal preferences among anglers for various preparation and cooking methods and other related habits (such as consumption of pan drippings). The assumption that there is no loss of PCBs during cooking or preparation, used in the RME point estimate risk calculations, is conservative, and may overestimate risks on average. The possible range of cooking losses was explicitly evaluated in the Monte Carlo analysis.

Exposure Point PCB Concentrations in Sediment and River Water. Exposure point concentrations for sediment and river water were calculated using the 20-year modeled data through 2018 (USEPA, 1999d). Although the exposure durations for recreators extend beyond the year 2018, concentrations for sediment and river water were not extrapolated to later years. This approach is conservative, since the concentrations are decreasing with time, and inclusion of later years would have resulted in lower concentrations. The concentration in sediment and water were not extrapolated because the concentration decline appears to be less than the decline in fish. In addition, although the upstream conditions are somewhat uncertain, the modeled concentrations assuming a constant-upstream boundary condition were adopted, although the choice of the boundary condition scenario has little impact on the model predictions (USEPA, 1999d).

Sediment Ingestion Rate. In the absence of site-specific ingestion rates, USEPA-recommended values for median daily soil ingestion were used in the risk assessment. The USEPA-recommended soil ingestion rates are somewhat uncertain. There is considerable debate in the scientific community regarding soil ingestion, and work is ongoing to better characterize soil ingestion rates. The soil ingestion rate exposure factor represents *total daily* intake of soil integrated over a variety of activities, including ingestion of indoor dust. In this HHRA, a median ingestion rate (as opposed to a high-end rate) was used for recreational exposures, because the total exposure time is only a fraction of the total day. The median ingestion rates used are likely high-end estimates of incidental sediment ingestion while participating in activities along the Hudson, because other sources (such as at home) also account for soil/sediment ingestion. On the other hand, increased dermal adherence of (wet) sediment compared to (dry) soil could correspond to higher actual ingestion rates for sediment than soil.

Sediment/skin adherence factor. This factor represents the amount of sediment that adheres to skin and is available for dermal exposure. Because this value is likely to vary based on one's activity, the values used for this parameter, which are estimates from single activities, are somewhat uncertain. For dermal contact with Upper Hudson River sediments, published adherence factors for adults gathering reeds, and for children playing in wet soils, were used as a surrogate for children. Although it is somewhat uncertain whether these scenarios are representative of contact with Hudson sediments, they appear to be a reasonable use of available data.

Dermal Absorption Value. The PCB dermal absorption rate used in this risk assessment was based on a value published in peer-reviewed literature. Nonetheless, since dermal absorption of soil and sediment contaminants is a complicated issue, there is considerable uncertainty associated with dermal absorption rates. Various factors affect the efficiency of dermal absorption. For example, many compounds are only absorbed through the skin after a long exposure duration (*i.e.*, >24 hours). Since most

individuals bathe at least once each day, washing may remove any soil residues adhering to the skin before absorption can occur. Therefore, dermal absorption rates based on studies with long exposure durations tend to overestimate actual absorption. However, soil loadings have also been shown to affect dermal absorption rates; the percentage of dermal absorption may increase as soil loadings decrease. The use of various testing methods also introduces uncertainties; *in vivo* animal studies introduce uncertainties regarding animal-to-human extrapolation, while *in vitro* studies using human skin introduce uncertainties regarding *in vitro* to *in vivo* extrapolations. Despite these uncertainties, the published dermal absorption values used in this risk assessment provide a reasonable basis to estimate risks for the dermal pathway.

PCB Concentrations in Air. The PCB concentrations in air used in this risk assessment are particularly uncertain, and the risks calculated for this pathway should therefore be considered to be "screening" level risks. Measurements of PCBs in air in 1991, adjusted to reflect the lower PCB concentrations in the water column at present and predicted into the future, provided one estimate for the exposure point concentration. These measurements were compared with modeled PCB volatilization and dispersion estimates. The two estimation methods provided a very wide range of concentration estimates. Despite the wide range of results, the results of the analysis indicate the volatilization of PCBs from the river is likely to yield *de minims* human health risks.

5.3.2 Toxicity Assessment

The toxicity values used in this risk assessment have been peer reviewed and are the most current values recommended by USEPA. The USEPA used uncertainty factors of up to 300 in deriving reference doses for Aroclor non-cancer assessment. Similarly, the PCB cancer slope factors were derived by USEPA using health protective dose-response models. These approaches may overestimate non-cancer hazards and cancer risks. Conversely, some uncertainties may lead to underestimation of cancer risks and non-cancer hazards. For example, Aroclors tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain, such as those found in the Hudson River.

The toxicity values used in the risk assessment are protective of both males and females. For example, the cancer slope factor used in calculating risks is based on an increased incidence of liver tumors in female rats reflecting the potential sensitivity of this gender. The slope factor generated based on female rats was higher than that generated for tumors found in male rats. Because risk is a function of exposure and hazard, the use of the higher slope factor based on data from the female rats is more protective of the general population than using the lower slope factor identified for male rats.

Although commercial PCBs tested in laboratory animals were not subject to prior selective retention of persistent congeners through the food chain, the CSFs are based on animal exposures to a group of PCB mixtures (*i.e.*, Aroclor 1260, 1254, 1242, and 1016) that contain overlapping groups of congeners spanning the range of congeners most often found in environmental mixtures.

One of the RfDs used in the risk assessment is based on several studies of monkeys where females were exposed through ingestion prenatally and as adults. The studies found reduced birth weights in offspring of the prenatally exposed monkeys and immune effects in adult female monkeys exposed for longer periods of time. The No Observed Adverse Effect Levels identified from these studies were further reduced by factors of 100 and 300 to account for extrapolation from animals to humans and for

sensitive human populations. Thus, the use of this RfD in assessing potential non-cancer health effects is considered to be health protective. More recent data (Arnold *et al.*, 1995; Rice, 1999) indicate that the margin of safety afforded by the current RfD may be smaller. It should be noted that USEPA is currently reassessing the toxicity criteria for non-cancer effects of PCBs.

Toxic Equivalency Factors (TEFs) for Dioxin-Like PCBs. There is considerable uncertainty regarding the TEF values for the toxicity of dioxin-like PCB congeners. In their publications, WHO indicates that their TEF values represent "*an order of magnitude estimate* of the toxicity of a compound relative to TCDD" (emphasis added) (Van den Berg *et al.*, 1998). Also, the TEF analysis assumes that the toxic effects of dioxin-like PCBs are additive. However, this assumption is somewhat uncertain. As discussed in the WHO/ICPS TEF reviews (Ahlborg *et al.*, 1994; Van den Berg *et al.*, 1998), although there is evidence of additivity for Ah receptor mediated responses, interactions between nondioxin-like PCBs and dioxin-like PCBs may be antagonistic, in which case the assumption of additivity is highly conservative. However, evidence of synergistic interactions also exists. It is also important to note that many nondioxin-like PCB congeners have independent mechanisms of toxicity (Hansen, 1998). Although the toxicity of these congeners is likely to be reflected in the toxicity values developed for Total PCBs, the toxicity of each PCB congener has not been fully characterized, and TEF values have not been developed for non-dioxin-like congeners.

Research into possible endocrine effects of PCBs is an area of active research to develop toxicological tests to evaluate possible endocrine disruption. Although PCBs may also act as an environmental endocrine disruptor, the available data are insufficient to support a quantitative assessment of endocrine effects in this risk assessment. As discussed in Section 4.4, it is recognized that this is a source of potential uncertainty. Many of the standard toxicity tests performed to date on PCBs were not specifically designed to identify effects of endocrine disruption, and some health endpoints could have been missed by those studies. However, the Technical Panel concluded, based on available evidence, that exposure to xenoestrogenic chemicals, at current environmental concentrations, is probably insufficient to evoke an adverse effect in adults (USEPA, 1997b). Additional information is required to understand the mechanism by which the endocrine effects are acting, and to determine if this holds for the human fetus and neonate.

5.3.3 Comparison of Point Estimate RME and Monte Carlo Results

Each of the uncertainties associated with the Exposure and Toxicity Assessment steps in the risk assessment process becomes incorporated into the risk estimates in the Risk Characterization step. A comparison of the central tendency and RME point estimate risks for fish ingestion, with the Monte Carlo estimates, provides a perspective on the variability and uncertainty in the range of risks possible for this pathway under a wide range of scenarios.

A sensitivity/uncertainty analysis consisting of 72 combinations of the important exposure variables for the fish ingestion pathway was performed for the Monte Carlo analysis. A comparison of the base case Monte Carlo results with the point estimate results was presented in Section 5.2. As that comparison showed, the RME cancer risk estimate (1.1×10^{-3}), falls somewhat above the 95^{th} percentile of the base case Monte Carlo distribution of risk.

Tables 5-38 and 5-39 provide a summary of the point estimate HI and cancer risk estimates together with the full range of Monte Carlo estimates. Figures 5-3a and 5-3b plot percentiles for all 72 combinations of the non-cancer HI values and the cancer risks, respectively. The central (50^{th} percentile) Monte Carlo HI ranges from a low of 1.8, to a high of 51.5, compared to the CT point estimate of 10. The high-end (95^{th} percentile) Monte Carlo HI ranges from 18.6 to 366, compared to the RME point estimate of 116. A similar comparison for cancer risk indicates the 50^{th} percentile cancer risk estimates range from 9.7×10^{-6} to 4.1×10^{-4}, compared to a CT point estimate of 3.2×10^{-5}. The 95^{th} percentile Monte Carlo cancer risk estimates range from 1.1×10^{-4} to 3.1×10^{-3}, compared to the RME point estimate of 1.1×10^{-3}.

A discussion of the sensitivity of the Monte Carlo results as a function of several important exposure factors follows.

Uncertainty in Fishing Locations. For the base case Monte Carlo analysis, and the point estimate analysis, PCB concentrations in fish were averaged over the three locations modeled: Thompson Island Pool (River Mile 189), Stillwater (River Mile 168), and the Waterford/Federal Dam area (average of River Miles 157-154). However, it is possible that an angler would preferentially fish in a single location. To address this possibility, the Monte Carlo analysis considered catching and consuming fish from the most contaminated and least contaminated locations.

As both the historical data and modeling results indicate, the PCB concentration in fish in the Upper Hudson River exhibits a declining concentration from upstream to downstream locations. Of the three locations modeled, Thompson Island Pool had the highest modeled PCB concentrations in fish. Holding all other exposure factors at their base case values, while assuming an angler catches and consumes fish exclusively from the upstream areas of the Upper Hudson River (using the Thompson Island Pool as a surrogate), yields the following estimates of non-cancer hazard and cancer risk:

Sensitivity Analysis-Fishing Location

Outcome	**Point Estimate[a]**	**Base Case Monte Carlo**	**High- End PCB Concentration (Thompson Is. Pool) - Monte Carlo[b]**
Non-Cancer HI			
Central Tendency (CT)	10	11	19
High-End (RME)	116	137	226
Cancer Risk			
Central Tendency (CT)	3.4×10^{-5}	6.4×10^{-5}	1.0×10^{-4}
High-End (RME)	1.1×10^{-3}	8.7×10^{-4}	1.5×10^{-3}

[a]Point Estimate values based on original exposure factors (unchanged).
[b]Refer to Run #4 in Appendix B.
Base case Monte Carlo = 50^{th} percentile; High-End Monte Carlo = 95^{th} percentile.

As this comparison shows, the Monte Carlo HI and cancer risk increase by approximately 1.7 over their corresponding base case values for this scenario. This ratio is slightly larger than the approximately 1.5-fold difference in the point estimate weighted PCB concentrations.

Fish Ingestion Rate. The point estimate and base case Monte Carlo used the 1991 New York Angler survey as the basis for fish ingestion rates. As described in Chapter 3, the New York Angler survey yielded higher estimates of fish ingestion than a number of other studies. The 1992 Maine Angler survey (Ebert *et al.*, 1993) yields the lowest estimate of fish ingestion of the studies examined. An examination of the non-cancer hazards and cancer risk using the Maine fish ingestion rates yields the following:

Sensitivity Analysis-Fish Ingestion Rate

Outcome	Point Estimate[a]	Base Case Monte Carlo	Using Maine Angler Study Fish Ingestion - Monte Carlo[b]
Non-Cancer HI			
Central Tendency (CT)	10	11	6
High-End (RME)	116	137	85
Cancer Risk			
Central Tendency (CT)	3.4×10^{-5}	6.4×10^{-5}	3.4×10^{-5}
High-End (RME)	1.1×10^{-3}	8.7×10^{-4}	5.2×10^{-4}

[a]Point Estimate values based on original exposure factors (unchanged).
[b]Refer to Run #28 in Appendix B.
Base case Monte Carlo = 50th percentile; High-End Monte Carlo = 95th percentile.

As this comparison shows, the Monte Carlo HI and cancer risk decrease by approximately 0.5 over their corresponding base case values for this scenario. This comparison indicates that adopting a lower estimate of the fish ingestion rate than the base case estimate does not change the results significantly.

Exposure Duration. The point estimate and base case Monte Carlo analysis defined exposure duration based on the joint distribution of residence duration and fishing duration. As a sensitivity analysis, residence duration alone was used to examine the non-cancer hazards and cancer risk under this scenario:

Sensitivity Analysis-Exposure Duration			
Outcome	**Point Estimate[a]**	**Base Case Monte Carlo**	**Exposure Duration based on Residence Duration Only - Monte Carlo[b]**
Non-Cancer HI			
Central Tendency (CT)	10	11	14
High-End (RME)	116	137	163
Cancer Risk			
Central Tendency (CT)	3.4×10^{-5}	6.4×10^{-5}	1.1×10^{-4}
High-End (RME)	1.1×10^{-3}	8.7×10^{-4}	1.4×10^{-3}

[a]Point Estimate values based on original exposure factors (unchanged).
[b]Refer to Run #37 in Appendix B.
Base case Monte Carlo = 50th percentile; High-End Monte Carlo = 95th percentile.

As this comparison shows, the Monte Carlo HI increases by approximately 1.2, and the cancer risk increases by approximately 1.6 over their corresponding base case values for this scenario. This comparison indicates that adopting a higher estimate of the exposure duration than the base case estimate does not change the results significantly.

Population Risks. Consistent with USEPA's Superfund guidance, this risk assessment does not estimate the number of anglers that consume their catch or the number of women of child-bearing age exposed through consumption of fish because CERCLA requires consideration of risk to an individual with a reasonable maximum exposure. It would be difficult to identify the number of anglers who are consuming fish in the presence of fishing bans and health advisories, because of the potential for underreporting and the threat of fines for anglers keeping fish from the Upper Hudson River. It is also not possible to project with any certainty the number of potential anglers within various stretches of the river who would consume fish if there were no health advisories in the Upper Hudson River.

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